IMMEDIATE OUTCOME AND RISK FACTORS DETERMINING THE OUTCOME OF STATUS EPILEPTICUS IN CHILDREN ATTENDING TERTIARY CARE CENTRE

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CERTIFICATE

This is to certify that the dissertation titled**, "IMMEDIATE OUTCOME AND RISK FACTORS DETERMINING THE OUTCOME OF STATUS EPILEPTICUS IN CHILDREN ATTENDING TERTIARY CARE CENTRE"** submitted by Dr.T.Murali, to the Faculty of Paediatrics, The Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfilment of the requirements for the award of M.D. Degree (Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance, during the academic year 2009-2011.

Dr. T.Kathir Subramaniyam. M.D.,D.C.H.,
Unit Chief,
Department Of paediatrics,
Tirunelveli Medical College,
Tirunelveli.

Dr.T.Kathir Subramaniyam. M.D.,D.C.H.,
Professor and Head,
Department Of Paediatrics,
Tirunelveli Medical College,
Trunelveli.

The Dean

Tirunelveli Medical College,

Tirunelveli.

DECLARATION

I, Dr.T.Murali, solemnly declare that the dissertation titled "**IMMEDIATE OUTCOME AND RISK FACTORS DETERMINING OUTCOME OF STATUS EPILEPTICUS IN CHILDREN ATTENDING TERTIARY CARE CENTRE"** has been conducted by me at Tirunelveli Medical college, Department of Pediatrics, Tirunelveli, during 2009-2011 under the guidance and supervision of my unit chief **PROF.T.KATHIR SUBRAMANIYAM,M.D,D.C.H.,** This is submitted in partial fulfilment of the award of the degree of M.D,(Pediatrics) for the April 2011 examination to be held under the Tamilnadu Dr. M.G.R. Medical University, Chennai .This study has not been submitted previously by me for the award of any degree or diploma from any other university.

(Dr.T.MURALI.)

M.D.Paediatric Medicine

Postgraduate Student

Tirunelveli Medical College

Tirunelveli

TIRUNELVELI MEDICAL COLLEGE AND HOSPITAL

TIRUNELVELI

INSTIUTIONAL ETHICAL COMMITTEE

CERTIFICATE OF APPROVAL

This is to certify that the INSTIUTIONAL EITHICAL COMMITTEE of TIRUNELVELI MEDICAL COLLEGE AND HOSPITAL, TIRUNELVELI -11 has unanimously approved the dissertation titled Clinical profile immediate outcome analysis of risk factor determining adverse outcome in children with status epilepticus by Dr. T. Murali, MD., Paediatrics Student, TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI -11 in its meeting held on 09.10.2010.

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Ethical Committee,
Tirunelveli Medical College,
Tirunelveli-11.

TIRUNELVELI MEDICAL COLLEGE
TAMILNADU
INDIA
TIRUNELVELI-11

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CONTENTS

INTRODUCTION

Status epilepticus (SE) is a paediatric and neurological medical emergency associated with significant morbidity and mortality. Status epilepticus represents the brains reaction to an acute insult or it may be a modification of already existing epilepsy. The mortality associated with SE in children is considerable without treatment. Hence optimum management includes prompt recognition, appropriate therapy, and investigations for the cause of status epilepticus followed by correction.

The incidence of status epilepticus has a bimodal distribution with peaks in children aged less than a year and the elderly.

SE is defined as a continuous seizure lasting more than 30 min, or two or more seizures without full recovery of consciousness between any of them.[1] However based on recent understanding of the pathophysiology, newer definition includes seizures that lasts more than 5 minutes needs to be treated as SE. If circulation is restored within 3-5 minutes, full recovery may occur, but if hypoxic-ischemia lasts beyond 3-5 minutes some degree of permanent cerebral damage is the rule. Status Epilepticus common in childhood, and the reported current mortality is in the range of 4-6% [2]. The evolution of a prolonged seizure into Status Epilepticus is associated with increased morbidity and mortality. Hypoxia is currently thought to be responsible for most of the complications seen in Status Epilepticus[3]. SE in children is a life-threatening condition with serious risk of neurological sequelae. The outcome from an episode of SE is determined primarily by its cause, but the duration of the seizure and prompt, appropriate treatment are still extremely important contributing factors. Any type of seizure can lead to status epilepticus, but generalized tonic- clonic status epilepticus is the most common and most dangerous type. The longer seizures continue, the more difficult they are to control and the higher the morbidity and mortality. Permanent neuronal damage can occur after 60 minutes of seizure activity.

STUDY JUSTIFICATION

Status epilepticus is one of the most common paediatric and neurological emergencies. Status epilepticus is an under-recognized health problem associated with substantial morbidity and mortality. Data on outcome in status epilepticus in children is sparse in our country.

Though we managed status epilepticus in our tertiary care level hospital we do come across poor outcome in SE. Mortality remain high due to longer duration of travel with prolonged SE, improper prehospital therapy, refractory seizures, etiological factors and risk factors influencing the poor outcome. This study done to identify these risk factors for both death and neurological sequelae.

If these determinant factors identified and managed aggressively and in a time bound manner, outcome of status epilepticus in children can be improved.

REVIEW OF LITERATURE

Status epilepticus (SE) has been defined as continuous seizure activity lasting more than 30 min (or) 2 or more seizures in this duration without regaining consciousness in between them. However, the operational definition has brought the time down to 5 min.

ETIOLOGY :

The etiological factors that causes SE and affect the outcome of SE are febrile seizures, acute infections, trauma, metabolic derangements, drug overdose, hypoxia, idiopathic epilepsy, drug withdrawal, structural brain damage, static causes, degenerative disorder etc.

ETIOLOGICAL CLASSIFICATION OF SE[4]

TYPE	DEFINITION	EXAMPLES
Remote symptomatic	SE occurring without an acute provocation in a patient with a history of CNS insult	CNS malformation, Previous traumatic brain injury or insult, chromosomal disorder
Acute symptomatic	SE occurring during an acute illness (e.g., acute CNS insult, acute encephalopathy)	Meningitis, encephalitis, electrolyte disturbance, hypoxia, intoxication, sepsis, trauma
Febrile	SE occurring when the only provocation is a febrile illness, after excluding a direct CNS infection	Upper respiratory infection, sinusitis, sepsis

Cryptogenic (Idiopathic)	SE occurring in the absence of an acute precipitating CNS insult, systemic metabolic disturbance, or both	No definable cause
Progressive encephalopathy	SE occurring with an underlying, progressive CNS disorder	Amino or organic acidopathies, CNS lipid storage diseases, mitochondrial disorders
Remote symptomatic with an acute precipitant	SE occurring with a chronic encephalopathy, but with an acute provocation	CNS malformation or previous CNS insult with concurrent infection, hypoglycemia, hypocalcemia, or intoxication

PATHOPHYSIOLOGY :

ACUTE SYSTEMIC AND CNS COMPLICATION:

Acute systemic effects of GTCS include cardiovascular alteration, acidosis, respiratory distress, hyperthermia and renal failure secondary to rabdomyolysis. The physiological alteration occurs in two phases.

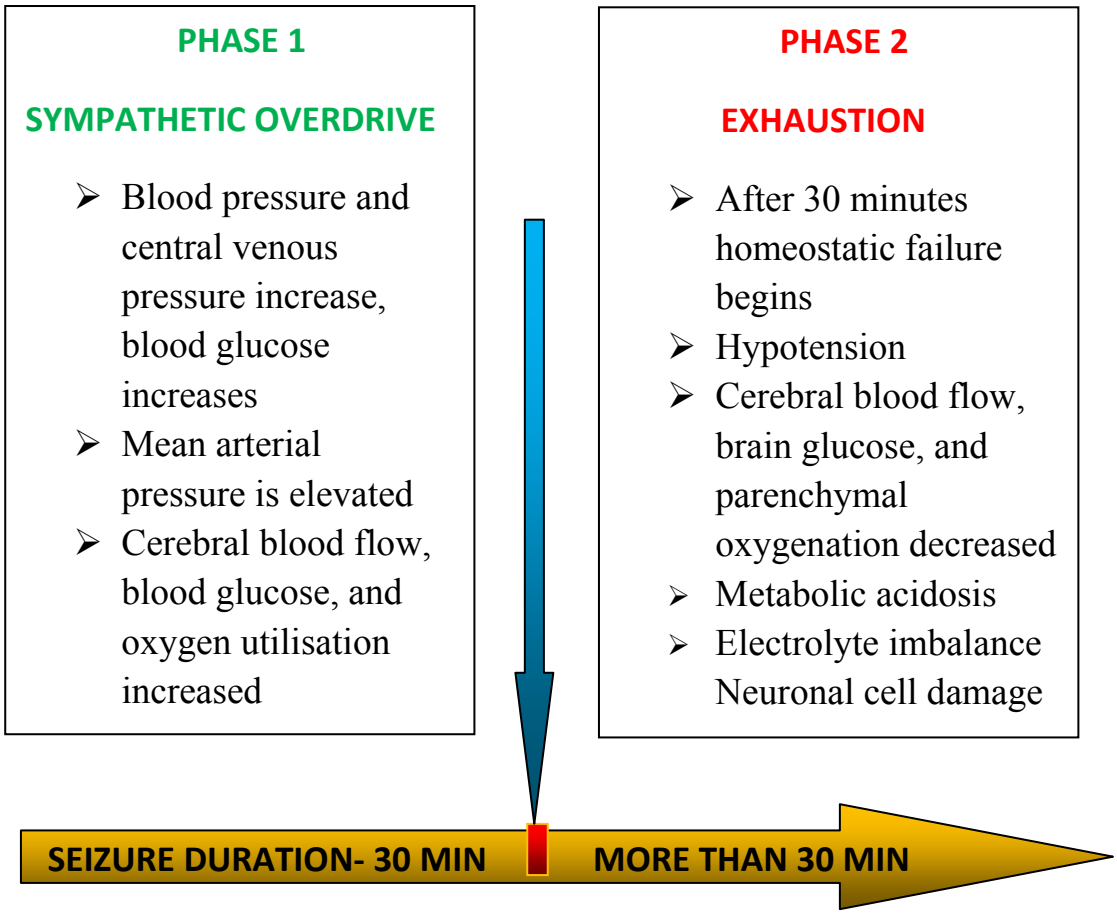
PHASE I

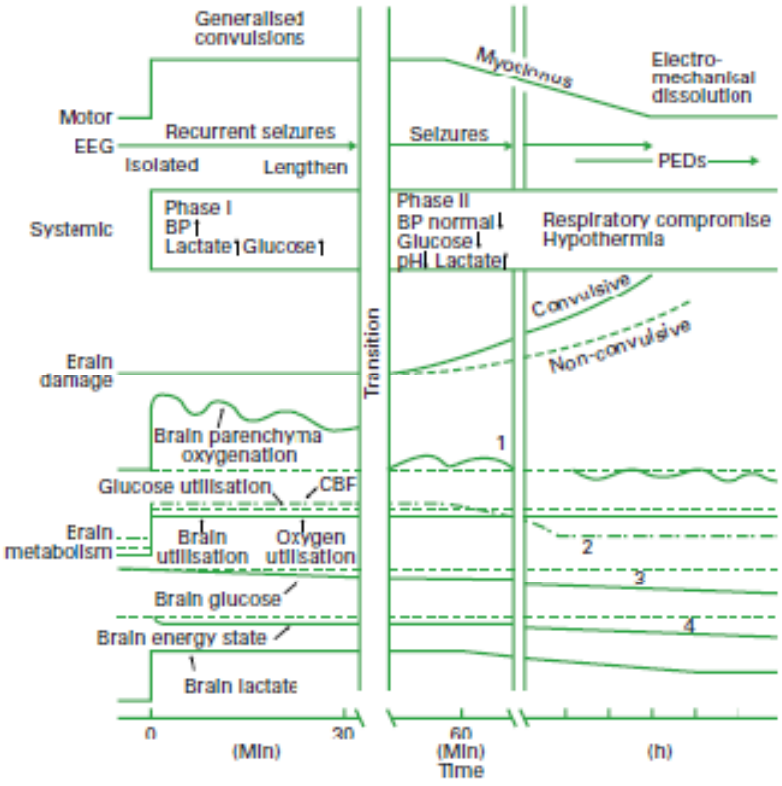
Initial phase last during the first 30 minutes of SE. This early phase is characterised by sympathetic overdrive. The systemic effects of CSE are initially dominated by the body's attempt to maintain homeostasis.[5] Blood pressure and central venous pressure increase, blood glucose increases, and the patient become tachycardic.[6] Mean arterial pressure is elevated as

a result of increased vascular resistance. CSE may also result in electrolyte imbalance and hyperthermia[7]. Cerebral blood flow, blood glucose, and oxygen utilisation increase in the initial phases of a seizure to maintain cerebral homeostasis.

PHASE II

After 30 minutes homeostatic failure begins and the patient may need systemic support. Cerebral blood flow, brain glucose, and parenchymal oxygenation all decrease and potentially play a part in the cell damage associated with CSE[6]. Respiratory and metabolic acidosis, electrolyte imbalance (for example, hyperkalemia), hyperthermia, and rhabdomyolysis may all occur .Treatment with drugs having depressant cardiorespiratory side effects (for example, benzodiazepines and barbiturates) may worsen the systemic complications of CSE.





Temporal changes which occur as tonic-clonic status epilepticus progresses. Motor activity lessens, the electroencephalogram (EEG) evolves and profound physiological changes occur, both systematically and cerebrally. In the first 30 minutes or so, physiological changes are largely compensatory, but as the seizures continue these compensatory mechanisms break down. The biphasic evolution is emphasised periodic epileptic discharge (PED). 1, loss of reactivity of brain oxygen tension; 2, mismatch between the sustained increase in oxygen and glucose utilisation and a fall in cerebral blood flow; 3, a depletion of cerebral glucose and glycogen concentrations; 4, a decline in cerebral energy state.

SEIZURE INITIATION AND PROLONGATION

Seizure initiation is caused by an imbalance between excitatory and inhibitory neurotransmission, leading to the initiation of abnormal neural impulses. The seizure threshold in the immature brain appears to be lower than in the mature brain, but the mechanisms that underlie this susceptibility remain unclear. Excitatory synapses mature

earlier than inhibitory synapses and this, coupled with an increase in the susceptibility of excitatory neurotransmitter receptors, increases the likelihood that an excitation–inhibition imbalance may occur[8 9].

There are other important differences between the immature and adult brain. Stimulation of $GABA_A$ receptors in the immature brain results in depolarisation rather than hyperpolarisation[10] The immature cerebral cortex has a high synaptic density at around 2 months of age and this may contribute to the development of hyper synchrony of neural groups[9].

The excitatory amino acid neurotransmitter glutamate increases at the site of the seizure focus at the beginning of seizure activity in adults with temporal lobe epilepsy when measured by in vivo intracerebral microdialysis[11]. It is believed that the same may happen at the onset of generalised seizures. Inhibitory neurotransmitters such as GABA later increase at the seizure focus and redress the balance between excitation and inhibition. GABA also increases in the substantia nigra, pars reticulata, an area that can modulate a cortical inhibitory response in adult rats, but not in immature rats.

Other mechanisms of inhibitory receptor modulation, such as adenosine receptor agonist, may also contribute to seizure termination.

ELECTROPHYSIOLOGY

CSE starts with localised epileptic activity followed by isolated generalised bursts of seizure activity with a normal EEG in between. If the patient does not regain consciousness between these episodes, then they meet the clinical criteria for CSE. The isolated ictal discharges merge and become a continuous discharge after about 30 minutes. Discharges then fragment and are interspersed with flat periods. Ultimately, periodic epileptiform discharges, which may reflect underlying metabolic failure, will occur.[5 12]

The motor phenomena associated with CSE follow a similar pattern to the EEG changes. Recurrent seizures will merge into continuous motor activity, followed by fragmentation of the motor activity and myoclonus. If the seizure persists, then electromechanical dissociation will ensue. The prognosis for a good neurological outcome decreases the further the patient moves through this continuum.

ROLE OF EXCITOTOXIC AMINO ACIDS IN THE DEVELOPMENT OF STRUCTURAL BRAIN DAMAGE SECONDARY TO CSE

Mesial temporal sclerosis is the most common acquired brain lesion following CSE and may result from excitotoxicity. They suggested that glutamate was directly responsible for the cell death, although the neurotransmitter role of glutamate was unknown. Since that time much animal model and cell culture work has attempted to prove this hypothesis and to relate it to status epilepticus.[13] Direct application of glutamate on to hippocampal cultures causes neuronal death.[14] This work provides indirect evidence that CSE can itself cause hippocampal damage.

CSE induces the production of heat shock proteins in several brain regions. The presence of heat shock proteins can protect the brain against further stressful stimuli, which are potentially damaging to neurones[15]. The implication is that prolonged seizures may need to occur in epilepsy human patients for mesial temporal sclerosis to develop, and that once it has developed further episodes of CSE may not worsen the mesial temporal sclerosis.

MECHANISMS BY WHICH GLUTAMATE CAUSES CELL DEATH

Excess extracellular glutamate may result in cell death by causing necrosis, gene determined cell death, or both. The primary receptor involved in cytotoxicity related to glutamate is the NMDA receptor, although other glutamate receptors may be involved.[16] The NMDA receptor is an ionotropic receptor. Binding of glutamate and glycine or d-serine to

appropriate sites on the receptor results in an influx of calcium through the ionophore. High intracellular calcium concentrations result in

- Activation of protein kinase C.
- Nitric oxide and free radical formation.
- Activation of phospholipase A_2.
- Activation of protease calpain I. Calpain inhibitors are partially neuroprotective. Glutamate receptor stimulation also results in the formation of immediate early genes, such as c-fos, fos-B, c-jun, and jun-B. c-fos encodes for Fos protein, which has a leucine zipper allowing it to bind and form dimers with similar proteins.[17] Some of the genes regulated are harmful and some are potentially neuroprotective.

The potentially toxic effects of metabotropic glutamate activation include the potentiation of NMDA and other excitatory membrane currents, the potentiation of intracellular calcium release, a decrease in inhibitory membrane currents, and decreased GABAergic inhibition. Conversely, potential protective effects include the inhibition of synaptic glutamate release and decreased calcium influx.[18]

Children who develop CSE in the neonatal period do not appear to develop mesial temporal sclerosis, but others are most vulnerable under the age of 3 years.

REFRACTORY STATUS EPILEPTICUS:

RSE is defined as seizures that are not controlled with adequate doses of BZD, Phenytoin ,phenobarbitone and require more aggressive treatment.[19]Children with prior, neurological problem, progressive CNS injury, complications, refractory status epilepticus have poor outcome. Continous IV infusion of midazolam, propofol, sodium valproate, thiopentone sodium are the most useful treatments

NONCONVULSIVE STATUS EPILEPTICUS:

This condition is recognised , when an unresponsive child presents with conjugate deviation of eyes, eyelid twitch, nystagmus or unilateral clonus. NCSE should be anticipated when a child is brought with history of seizures but has not regained baseline consciousness and does not have obvious convulsive activity.A high index of suspicion is needed to identify the NCSE during the course of management of CSE. Convulsive activity disappears but the child remains apneic or tachypneic, shocky and unresponsive. The eye signs indicate the persistence of ongoing seizure activity. Continuing aggressive management of the airway breathing and circulation with the same drug protocol of CSE will improve the outcome.

TYPES OF STATUS EPILEPTICUS

GCSE/ GTCS (primary)

Secondary generalised

Multifocal clonic

Hemiconvulsive SE

Tonic SE

Clonic SE

Simple PSE/epilepsia partia continualis

Non convulsive status epilepticus(Subtle SE)

Neurological EMD(electrical SE)

PCSE

ASE

Myoclonic SE+/- Salam attacks

Complications of status epilepticus [20]:

1. Interictal coma.
2. Cumulative anoxia- cerebral and systemic.

3. Cardiovascular complications – Tachycardia, Bradycardia, cardiac arrest, hypertension, cardiac failure, shock.
4. Respiratory system failure- Apnea, Cheyne-stokes breathing, aspiration, pneumonia, tachypnea, neurogenic pulmonary oedema, pulmonary embolism, cyanosis, and respiratory acidosis.
5. Renal failure- Oliguria, uremia, acute tubular necrosis, rhabdomyolysis.
6. Autonomic disturbances – Hyperpyrexia, sweating, vomiting, hyper secretion, airway obstruction.
7. Metabolic and Biochemical abnormalities – Acidosis (Metabolic, lactic acidosis), hyper and hyponatremia, hyperkalemia, hypoglycemia, hepatic failure, dehydration, acute pancreatitis.
8. Infections- pulmonary, bladder.
9. Others – Disseminated intravascular coagulation, multiple organ dysfunction, fractures, and thrombophlebitis.

INVESTIGATIONS

Complete blood count

Blood sugar

Serum calcium, electrolytes,

CXR

CT Brain /USG Cranium

CSF analysis

EEG,

X-ray skull

MRI

Renal function test

Liver function test

Serum magnesium

Serum ammonia, lactate

ABG

Blood culture and sensitivity

Toxicological screening

AED Level in blood /urine

Metabolic screening

EEG

In a child with a new onset of seizures, an EEG may help to differentiate ictal from non ictal events, to determine seizure type or epilepsy syndrome and to better define the risk for recurrence. For most children it is not necessary to perform the EEG as part of the initial emergency department evaluation. In fact if it is performed shortly after the seizure (<48 hours) the EEG may show diffuse post ictal slowing without prognostic significance. Among children with persistent altered mental status after a seizure an emergent EEG is helpful to identify subtle or NCSE.

NEURO IMAGING: CT Brain may be necessary to evaluate safety of LP and to rule out haemorrhage or large mass lesions. MRI will almost always be performed later, even if CT is normal.

Lumbar Puncture should be done if SE presented in febrile children. And it should be done only after stabilizing the child not at arrival.

MANAGEMENT:

Status epilepticus is a medical emergency that requires an organised and skillful approach to minimize the associated mortality and morbidity. longer the duration of seizures, greater the risk of complications. Hence protocol based therapy must be aggressive because

neuronal excitability can be reversed only early in the course and quick intervention may decrease the risk of seizures generated neuronal damage.

CSE is an emergency and the fact that aetiology emerges as the most powerful predictor of outcome should not be read as meaning that interventions may not alter that outcome. The main objectives of treatment, which should be addressed simultaneously, are:

a) Support of vital functions and oxygenation.

b) Termination of seizure .

c) Identification and treatment of causal or precipitating factors.

a) SUPPORT OF VITAL FUNCTIONS AND OXYGENATION

Treatment should follow the ABC principles of resuscitation in order to maintain airway, ventilation-oxygenation and blood pressure.

AIRWAY AND BREATHING

Open the airway using the head tilt and chin lift maneuver. C-spine precaution are taken if trauma is suspected gentle oropharangeal suction done .Oxygen is provided with the non-rebreathing mask .If the patient is not breathing a bag and mask ventilation is used to assist ventilation. If the patient is not ventilating due to convulsion or is become cyanosed, the airway is secured using rapid sequential intubation technique. Continuous monitoring of vital signs, ECG and pulse oximetry are recommended. The airway, breathing and circulation take precedence over seizure control. Intermittent or persistent apnea is not a contraindication for AED but an indication for establishing airway and breathing.

GLUCOSE

Hypoglycemia can cause severe disruption of autoregulation of cerebral blood flow.Patient should have prompt measurement of blood glucose with the help of dextrostix .If hypoglycemia is documented or it is not possible to measure blood glucose a bolus of 2ml/kg of 25% dextrose solution, may be given intravenously.

CIRCULATION

Within the first ½ hour of SE, blood pressure rises. Later, blood pressure either becomes normal or hypotensive. The circulatory shock which follows ongoing SE, severely deranges cerebral physiology. Hence it is necessary to monitor heart rate, peripheral perfusion and BP. Correct shock if identified with isotonic fluids.

b)TERMINATION OF SEIZURE

Rapid treatment of status epilepticus is crucial to prevent neurologic and systemic pathology. The goal of treatment always should be immediate diagnosis and termination of seizures. It is believed that early pharmacological intervention leads to termination of seizures with smaller doses than would be required if seizures were allowed to progress[21]. During status epilepticus there will be time-dependent loss of synaptic GABAA receptors and thus of GABA-mediated inhibition, which correlates with the progressive pharmacological resistance to GABAergic medication observed in refractory CSE.[22 23]

BENZODIAZEPINES

The benzodiazepines are some of the most effective drugs in the treatment of acute seizures and status epilepticus. The benzodiazepines most commonly used to treat status epilepticus are diazepam, lorazepam , and midazolam. All three compounds work by enhancing the inhibition of γ-aminobutyric acid (GABA) by binding to the benzodiazepine-GABA and barbiturate-receptor complex.

DIAZEPAM

Diazepam is one of the drug of choice for first-line management of status epilepticus[24] [25] Dosage:0.05-0.3mg/kg IV, at the rate of 0.1mg/kg/min Although the drug enters the brain rapidly because of its high lipid solubility, after 15 to 20 minutes it redistributes to other areas of the body, reducing its clinical effect[26] [27] Despite its fast distribution half-life, the

elimination half-life is approximately 24 hours. Thus, sedative effects potentially could accumulate with repeated administration.

Diazepam in a typical intravenous dosage of 5 to 10 mg per minute terminates seizures of any type in about 75 percent of patients with status epilepticus[28]. Adverse effects include respiratory suppression, hypotension, sedation, and local tissue irritation. Diazepam also may be given rectally.

Despite its pharmacokinetic and adverse effect limitations, diazepam remains an important tool in the management of status epilepticus because of its rapid and broad-spectrum effect.

LORAZEPAM

Lorazepam has emerged as the preferred benzodiazepine for acute management of status epilepticus. Lorazepam differs from diazepam in two important respects. It is less lipid-soluble than diazepam, with a distribution half-life of two to three hours versus 15 minutes for diazepam.Onset of action of lorazepam is 1-3 min. Therefore, it should have a longer duration of clinical effect. The anticonvulsant effects of lorazepam last 6 to 12 hours, and the typical dose ranges from 0.05mg/kg to 0.3mg/kg IV. This agent also has a broad spectrum of efficacy, terminating seizures in 75 to 80 percent of cases. Thus, lorazepam also is an effective choice for acute seizure management, with the added possibility of a longer duration of action than diazepam.

MIDAZOLAM

Midazolam has no advantage over diazepam or lorazepam.dosage:0.1-0.15mg/kg IV, with onset of action 1-5 min.It is extremely efficacious as an intra muscular anti convulsant when other routes are not available.So ideal for pre hospital therapy.It has rapid onset of action and controls seizures in 90% of patients.Its shorter half life and resultant increased risk of recurrence makes it a less preferred drug to lorazepam in the initial management of CSE.

A second dose of lorazepam or diazepam may be repeated in 5 - 10 min if seizures are not controlled with the first dose.

PHENYTOIN

Phenytoin is one of the most effective drugs for treating status epilepticus, not responding to the initial 2 doses of benzodiazepines. It is given as 20mg/kg IV over 20 min.Then if needed,10mg/kg iv infusion over 20 min.[29]

The main advantage of phenytoin is the lack of a sedating effect. Side effects like arrhythmias and hypotension, local irritation, phlebitis, and dizziness may accompany intravenous administration.

FOSPHENYTOIN

Fosphenytoin is a water-soluble pro-drug of phenytoin that completely converts to phenytoin following parenteral administration within 8 to 15 minutes.[30] [31] It is metabolized by the liver and has a half-life of 14 hours. Because 1.5 mg of fosphenytoin is equivalent to 1 mg of phenytoin.The initial dose of fosphenytoin is 15 to 20 mg PE per kg, so it can be infused at a rate as high as 150 mg PE per minute, a rate of infusion that is three times faster than that of intravenous phenytoin. Intramuscular doses also can be given, but the drug does not reach a therapeutic level for 30 minutes.[32]

Adverse effects that are unique to fosphenytoin include perineal paresthesias and pruritus; however, both are related to higher rates of administration.[33] Unlike phenytoin, fosphenytoin does not cause local irritation. Intravenous therapy has been associated with hypotension, so continuous cardiac and blood pressure monitoring are recommended.

Intravenous pyridoxine 100 mg should be administered to all children below two years with cryptogenic CSE, not responded to phenytoin infusion. Diagnosis of pyridoxine-dependent seizures is not to be missed.[34], Apnea is a complication during administration.

PHENOBARBITAL

Phenobarbital typically is used after a benzodiazepine or phenytoin has failed to control status epilepticus. The normal loading dose is 15 to 20 mg per kg over 30 minutes. Because high-dose phenobarbital is sedating, airway protection is an important consideration, and aspiration is a major concern. Intravenous phenobarbital also is associated with systemic hypotension.

MANAGEMENT OF REFRACTORY STATUS EPILEPTICUS

Seizures not controlled with adequate doses of benzodiazepines,phenytoin or phenobarbitone has been defined as refractory SE.

Midazolam appears to be a good choice for initial treatment of RSE in children. Midazolam is given as 0.15mg/kg IV bolus,followed by a continuous infusion of 1μg/kg/min with increments of 1μg/kg/min every 15 minutes until the seizure is controlled. The maximum rate of infusion is 15-20 μg/kg/min.The drug has anxiolytic, muscle relaxant, hypnotic and anti convulsant effects.

Status epilepticus not responding to midazolam, may be treated with sodium valproate,15-20mg/kg bolus (max 40mg/kg), followed by an infusion of 5mg/kg/hr. The advantages of this drug are that it has less sedation than other drugs, good cardiovascular profile and enables quick recovery.

A loading dose of thiopentone sodium 3-5mg/kg followed by an infusion has also been given for refractory status epilepticus. But severe hypotension requiring vasopressor therapy and prolonged post-infusion weakness delaying weaning make it a less commonly used drug in RSE.

Propofol has also been used in refractory status epilepticus in children. Loading dose:1-3 mg/kg and infusion:2-10mg/kg. Apnea, bradycardia, hypotension are some of the common adverse effects. However recovery is quick after cessation of therapy. During

administration of the drugs mentioned for RSE, EEG monitoring is mandatory in the intensive care unit.

c) IDENTIFICATION AND TREATMENT OF CAUSAL OR PRECIPITATING FACTORS

Management of CSE includes identification and treatment of the underlying cause. In the absence of an obvious aetiology, blood glucose, blood gases and electrolytes (including sodium, calcium and magnesium) should be tested and any metabolic derangement should be corrected. However, the diagnostic assessment should not delay treatment and clinicians should be able to diagnose CSE on clinical grounds alone. In general, an EEG, neuroimaging or other laboratory studies are not needed before the initiation of anticonvulsant therapy. If available, indications for emergency EEG include:[35]

- Clinical impression of pseudoseizures presenting as CSE.
- Unexplained altered awareness or no improvement or return to baseline of mental status after controlling overt convulsive movements (to exclude ongoing seizure electroencephalographic activity).
- Neuromuscular paralysis.
- High-dose suppressive therapy for refractory CSE.

OUTCOME

Outcome is determined by, etiology, age, duration, treatment. Ultimately mortality related to, damage to the CNS caused by the acute insult precipitating SE, systemic stress from SE(major cause because of anoxia, acidosis, shock), injury from repetitive epileptic discharges within the CNS. Mortality increases from 3% to 32% if the duration of seizures becomes more than 1 hour.

Neurologically normal children and children with febrile status have a favourable prognosis. Improved outcome is a result of timely and approximate evaluation and treatment.

Most favourable for patient who respond to first line agents, but obviously the underlying cause of status, determine the outcome. Cognitive function may be impaired (particularly memory) in patients with prolonged SE and is more common when significant hypoxemia(aspiration) intervened. Outcome may be worse when SE is managed inappropriately. Most common mistakes seen are, inadequate dosing, Failure to order maintenance therapy. Failure to do the latter results in recurrence. AED should be continued particularly if a structural lesion resulted in SE.

Gulati S,et al[36],Department of Pediatrics, AIIMS, New Delhi, India, done a retrospective study on the clinical profile, immediate outcome and possible risk factors of SE in pediatric age group admitted to PICU in a tertiary care center.Case records of 451 neuroemergency patients admitted between January 1993 to April 2000 analysed, out of which 30 patients had status epilepticus. The age group varied from 1 to 120 months with mean of 56.6+/- 46.5 months. Seventeen patients were less than 60 months. Sixteen patients (53.3%) presented with SE as first presentation without prior history of seizure activity. Nine patients died (30%) during hospital course. Seizure duration> 45 minutes (p-0.001) and presence of septic shock (p-0.001) were associated with significantly more mortality. This study conclude that there is a need to abort seizure activity at the earliest and this improves immediate outcome.

In an Indian study done by **Singhi S, Murthy A et al.**[37]**, at Chandigarh,** they compared the efficacy of continuous midazolam versus diazepam infusion in RSE. It was an open-label, randomized control study at their Intensive Care Unit. The subjects included 40 children, 2 to 12 years of age with refractory status epilepticus. They found that the median time to control seizures was 16 minutes in both the groups, but in the midazolam group, seizures recurred in more children (57% versus 16% in diazepam group; $p<0.05$). About half of the patients needed mechanical ventilation and 40% had hypotension in both groups but they found that the mortality was higher in the midazolam group (38%) as compared to the diazepam group (10.5 %, $p< 0.01>0.05$). They concluded that continuous midazolam and

diazepam infusions were equally effective for control of RSE. However, midazolam was associated with more seizure recurrence and higher mortality in RSE predominantly caused by central nervous system infections.

Rod C Scott et al,[38] Institute of Child Health, London,justified separate analysis in children as It differs from adult CSE in aspects of epidemiology, pathophysiology and clinical presentations. This study conclude that Febrile and acute symptomatic CSE are most common in children aged <2 years, whereas cryptogenic-idiopathic and remote symptomatic aetiologies are more common in older children. Mortality is 3-5% and morbidity directly attributable to CSE is less than 15%. Aetiology is the main determinant of outcome, while the separate effect on outcome of duration, age and treatment remains controversial. The risk of sequelae in unprovoked and febrile CSE is low.Prehospital administration of benzodiazepines is safe and simplifies subsequent management of CSE in the hospital setting. Treatment includes resuscitation measures, identification and treatment of causal factors and early antiepileptic treatment following local guidelines that may be based on national guidelines.

Sachin Admuthe,et al,[39] Retrospective study of clinical profile of Status Epilepticus in children.Institute of Child Health & Research Centre, Bijapur. from Jan 2004 to Dec2004, 27 patients who presented with status epilepticus. Their age group ranged from 4 months to 13 years with a mean of 4.5, 3.69 years. Boys and girls were almost in equal proportion (15:12). 10 patients (37.04%) with status epilepticus had previous history of seizure activity. The remaining 17 (62.96%) presented with status epilepticus as the first. Generalized Status Epilepticus is the most common type. The majority of children presented with Status Epilepticus at the first presentation are without a prior history of seizure activity.Most children with Status Epilepticus respond to combination of Midazolam and Phenytoin.

In a small retrospective study comprising of seven patients with refractory status epilepticus done by **Kumar A, Bleck TP** [40]**,** they found that midazolam infusions terminated

status epilepticus in all patients in less than 100 seconds as determined by clinical observation (three patients) or electroencephalographic monitory (four patients). But all the patients received mechanical ventilation before receiving midazolam. They found that one patient developed mild hypotension. In this small study, they concluded that midazolam appears to be an effective and safe alternative to high dose barbiturate coma for the termination of status epilepticus when conventional agents have failed.

Eriksson KJ, Koivikko MJ.[41]Tampere University Hospital, Finland done retrospective study includes 65 children treated for status epilepticus . Aetiology of the condition, effectiveness of the treatment protocol, including short barbiturate anaesthesia to prevent prolonged status epilepticus episodes, and neurological outcome were evaluated.There were no status epilepticus-related deaths. The cut-off point of status epilepticus duration for significant risk for permanent neurological sequelae was 2 hours. Treatment protocol, including short barbiturate anaesthesia in refractory cases, was able to abort status epilepticus in less than 2 hours in 75% of cases. This study conclude that early and prompt use of barbiturate anaesthesia should be encouraged, and may explain our low morbidity figures.

B G R Neville, R F M Chin, R C Scott[42] . Febrile CSE is the commonest single group with a good prognosis in sharp distinction to CSE related to central nervous system infections which have a high mortality. The aim of treatment is to intervene at 5 min and studies indicate that intravenous (i.v.) lorazepam may be a better first-line treatment than rectal diazepam and i.v. phenytoin a better second-line treatment than rectal paraldehyde. An epidemiological study strongly supports the development of prehospital treatment with buccal midazolam becoming a widely used but unlicensed option in the community. More than two doses of benzodiazepines increase the rate of respiratory depression without obvious benefit. The 1 year recurrence rate is 17% and the hospital mortality is about 3%.

Nicholas S Abend, Dennis J Dlugos[43] in their study aimed to describe nonconvulsive status epilepticus in terms of patient age, etiology, initial presentation, and electroencephalogram and neuroimaging findings. Twenty children with nonconvulsive status epilepticus were identified by a retrospective review of children who underwent long-term electroencephalogram monitoring in a pediatric intensive care unit. Age ranged from 2 months to 18 years, and in Nonconvulsive status epilepticus occurs in a heterogeneous group of children, results from acute symptomatic etiologies in children aged <1 year, most frequently follows isolated convulsions but can occur with only preceding mental status change, and is often prolonged. These findings suggest that a high level of suspicion for nonconvulsive status epilepticus must be maintained, and long-term electroencephalogram monitoring may be indicated in a large number of patients.

Gulser Esen Besli et al turkey.[44]study to evaluate the etiology, clinical profile, and short-term outcome of status epilepticus cases .Study group: 56 cases of SE who ages between 3 months and 15 years. Observed clinical course and outcome of 53 cases for 6 to 18 months. Results: While the most common cause of status epilepticus in children younger than 2 years is febrile, idiopathic/cryptogenic and remote symptomatic are in children older than 2 years. The rate of recurrence of seizure was significantly higher in cases with existing neurological abnormalities, prior epilepsy and seizures with remote symptomatic etiology. The most common triggering factors of SE development in cases with epilepsy were noncompliance for antiepileptic drugs and infectious fever. Conclusions: The risk factors of seizure recurrence were the presence of prior epilepsy, existence of neurological abnormalities and remote symptomatic etiology. Improving the compliance of patients and their families to take medicine appropriately and training them how to cope with febrile illnesses may decrease the recurrence of seizures.

Eriksson.K, et al,[45]contributing to the duration of a single convulsive seizure >5 minutes were analyzed in 157 children. The medically treated episodes were compared with seizure episodes resolving without treatment (n = 27). Major differences were in age (p = 0.016) and etiology (p = 0.003), and the association between treatment delay and response became significant after 30 minutes when this was analyzed as a single variable (p = 0.003) in Cox regression analysis.

DC Hesdorffer, G et al,[46] Mailman School of Public Health, Columbia University. studyTo determine the risk of recurrence of status epilepticus (SE) in a population-based sample and to identify risk factors for recurrence.All first episodes of afebrile SE between January 1, 1965, and December 31, 1984 included. Among the 183 episodes of first afebrile SE, the risk of recurrent SE was 31.7% over a 10-year follow-up period. The risk of recurrence was about 25% for those with acute symptomatic SE, remote symptomatic SE, and idiopathic cryptogenic SE. Recurrence was 100% for those with progressive symptomatic SE. Female gender and progressive symptomatic etiology increased the risk for recurrent SE. Study shows that Status epilepticus (SE) recurs in about one-third of individuals with a first episode of SE. Except for SE occurring in the setting a progressive brain disorder, the risk of recurrence is about 25%, regardless of the underlying etiology. Female gender and lack of response to the first antiepileptic drug medication are at greatest risk for recurrence.

R K Singh S et al[47]_Washington, DC. Prospectively study a total of 1,382 patients presented with new-onset seizures between 2001 and 2007 to characterize children with new-onset seizures presenting as status epilepticus at a tertiary care children's hospital. The average age was 3.4 years. The majority of seizures (72%) lasted between 21 and 60 minutes, had no significant past medical history; one-fourth had a family history of epilepsy. Combined CT and MRI provided a diagnosis in 30%. CT was helpful in identifying acute vascular lesions and acute edema, whereas MRI was superior in identifying subtle

abnormalities and remote symptomatic etiologies such as dysplasia and mesial temporal sclerosis.Conclude that Children who present in status epilepticus that is not a prolonged febrile convulsion should undergo neuroimaging in the initial evaluation. For any child who presents in status epilepticus and has not yet returned to baseline, the possibility of nonconvulsive status epilepticus should be considered. Although CT is often more widely accepted, especially in the urgent setting, strong consideration for MRI should be given when available, due to the superior yield.

Lacroix J, et al.[48]Canada.Retrospective study **t**o characterize the etiology, course, and prognosis in children admitted to a pediatric intensive care unit (ICU) for status epilepticus. One hundred forty-seven children. Status epilepticus was caused most often by epilepsy (n = 52), atypical febrile convulsions (n = 21), bacterial meningitis (n = 20), encephalitis (n = 20), intoxication (n = 8), or a metabolic disorder (n = 12). A normal neurologic status before status epilepticus and age < 4 yrs seem to be markers of good prognosis, while encephalitis and meningitis appear to be markers for morbidity and mortality. This study shows that most cases of status epilepticus were caused by epilepsy, atypical febrile seizure, encephalitis, meningitis, or metabolic disease. The mortality rate during the ICU stay was 6%. The prognosis was good in most surviving cases, more so if the neurologic development of the child was normal before the status epileptic.

Tirupathi S, et al[49].ireland Study is to identify clinical features and therapeutic decisions that influence admission, children presenting with CSE. In this study evaluated 47 cases over a three year period (2003-2006). Median age at presentation in the ICU group was 17 months (range 3 months-11 years) compared to 46 months in the ward group (range 3 months-10 years). Fifty per cent of patients in both groups had a previous history of seizures. Median duration of pre-hospital seizure activity was 30 min in both groups. ward. Febrile seizures were the most common aetiology in both groups.In this they conclude that younger

age at presentation, administration of more than two doses of benzodiazepines and deviation from the CSE protocol appear to be factors which influence admission of children to ICU. Recognition of pre-hospital administration of benzodiazepines and adherence to therapeutic guidelines may reduce the need for ventilatory support in this group.

. **Alldredge BK et al**,[50] This study showed that prehospital diazepam therapy was associated with SE of shorter duration (32 min vs 60 min; P = .007) and a reduced likelihood of recurrent seizures in the emergency department (58% vs 85%; P = .045). There were no significant differences between rectal and intravenous diazepam therapy with regard to SE duration, intubation, or recurrent seizures in the emergency department. These data suggest that prehospital administration of diazepam may shorten the duration of SE in children and simplify the subsequent management of these patients in the emergency department.

Towne AR, et al[51]. Determinants of mortality in status epilepticus. Epilepsia. 1994 Jan-Feb;35(1):27-34.

Using univariate and multivariate regression analysis ,they studied seizure duration, seizure type, age, etiologies, other clinical features, and mortality among 253 adults with status epilepticus (SE) admitted to the Medical College of Virginia. Cerebral vascular disease and discontinuation of antiepileptic drugs (AEDs) were the most prominent causes of SE, each accounting for approximately 22% of all patients in the series. The other principle etiologies were alcohol withdrawal, idiopathic, anoxia, metabolic disorders, hemorrhage, infection, tumor, drug overdose, and trauma. When the patients were divided into two groups, the group with SE lasting < 1 h had a lower mortality as compared with seizure duration > or = 1 h. Low mortality rates were noted in alcohol and AED discontinuation etiologies. Anoxia and increasing age were significantly correlated with higher mortality. The mortality rates of partial and generalized SE were not significantly different. Race and sex did not affect mortality significantly. Our findings represent the first multivariate analysis of predictive

indicators of mortality in SE and demonstrate that specific factors influence mortality rate in SE.

Stefan Beyenburg, et al,[52] study concludes that Although prospective studies are still lacking, NCSE may be one of the most frequently missed diagnoses in patients presenting with altered mental status. Elderly patients are at particular risk of diagnostic errors because of the broad range of presentations of NCSE, significant comorbidities (especially cerebrovascular disease), limited awareness of this particular seizure emergency or difficulties with access to electroencephalography. Although diagnostic criteria and treatment remain controversial, the diagnosis of NCSE is important because it is potentially reversible.

AIM AND OBJECTIVE

Aim of the study is to determine immediate outcome of status epilepticus in children in our hospital.

Secondary aim is to identify the risk factors influencing the outcome of status epilepticus

MATERIALS AND METHODOLOGY

STUDY DESIGN : Descriptive study

STUDY PERIOD : October 2009 to october 2010

STUDY POPULATION : Children from 1 month to12 yrs of age, who have been managed as SE

STUDY PLACE : Dept of Pediatrics, TVMC

INCLUSION CRITERIA : All children presented with SE (including non convulsive SE and secondary generalized), managed with anti convulsant as per protocol in the above age group in E.R.

EXCLUSION CRITERIA : seizures controlled before arrival to the hospital or before starting IV therapy.

Simple partial SE and myoclonic SE, with normal vital signs and without loss of consciousness Seizures occurred during hospital stay, not at arrival.

SAMPLE SIZE: All cases presented with status epilepticus in our pediatric casuality during the study period.

SAMPLING TECHNIQUE: All cases included over that period.

DROP OUTS: Totally 92 cases were included. Five cases absconded during hospitalisation.

SE: Defined as seizure lasting more than 30 min of a) continuous seizures or b) two or discrete seizures between which there is incomplete recovery of consciousness.

CSE: Refers to GTCS either primary or secondary to focal onset, in which whole or part of the body muscles having visible convulsions.

NCSE: refers to persistent seizure activity but no visible convulsions. Here they may have subtle signs of seizure activity such as unresponsiveness/ALOC or acute confusion state, apnea, defective DEM ,deviation of eyes, nystagmus.

NEURO EMD (ELECTRICAL SEIZURES): No visible sign of seizure activity, but EEG shows ongoing seizure activity. Example ; SE in patients paralysed with neuromuscular blockade.

INITIAL SEIZURE CONTROL; Seizure control by drugs at the time of first contact with the Emergency room.

CLINICAL CONTROL: No evidence of seizure activity such as convulsions, apnea, unresponsiveness, defective DEM, conjugate deviation of eyes, nystagmus, twitching of eyelids, lip smacking movements ,dilated pupils, tachycardia, excessive secretions, hypertension, airway stable with intact protective reflexes, stable respiration, DEM present ,with or without regaining consciousness.

GOOD RESPONSE: Defined as seizure activity controlled with AED within 60 minutes of contact with hospital and initiation of therapy .That is controlled with first line drugs.

REFRACTORY SE: RSE is defined as seizures that are not controlled with adequate doses of BZD, Phenytoin, phenobarbitone, and or seizure lasting more than 1 hour.

MORTALITY: Defined as death occurring in hospital during the course of treatment of SE without improvement and regaining baseline consciousness.

AMA : Means, discharged against medical advice during the treatment of status epilepticus without improvement and regaining baseline consciousness.

DURATION: Time starting from the onset of an episode of seizure prior till reaching the hospital or starting from the onset of first episode of seizure if having more than one episode of seizure with impaired level of consciousness.

HISTORY OF EPILEPSY: Defined as two or more unprovoked seizures in the past whether on treatment or not.

FEBRILE CSE: Status epilepticus occurring when the only provocation is a febrile illness, after excluding a direct CNS infection

MANOEUVRE:

92 children between the age group of 1 month to 12 years admitted in our hospital with the diagnosis of status epilepticus were included in the study group. Institutional consent and parental consent were obtained. First the cases were selected as per the inclusion criteria. Each child had been assessed on arrival and a preliminary history was obtained and documented in a preformed profoma. Rapid cardio pulmonary assessment was made, Spo2 on arrival, BP, presence of shock, pupil, were monitored. Before starting IV therapy, blood sample was taken for baseline investigations (sugar, calcium, and electrolytes).Then the cases were managed according to the protocol followed in our ER and admitted in wards/PICU.

Detailed history were obtained including duration of seizure, distance from the place where the fits occurred, mode of transport, prehospital therapy, precipitating factors, prior seizures/SE, drug history and compliance development milestone, prior neurological status. Complete clinical examination was made including neurological examination. Relevant investigation were done like CSF analysis CT brain /USG cranium, EEG, etc. during course of illness and therapy were monitored regularly till discharge. Computed tomography of brain was done in all patients with new onset of seizure and focal seizure. Magnetic resonant imaging of brain was done in selected cases.

Final diagnosis of various underlying problems was made based on the clinical history, physical examination and various investigation. The patients were further followed up for any complications acquired during hospital stay, duration of ventilation required length of stay in hospital stay. The neurological status of the child at the time of discharge was

noted. Time taken for control of refractory SE, time taken for full recovery of consciousness, requirement for prolonged ventilator support ,refractory shock or subsequent shock following midazolam or due to complication like sepsis, maximum dose and duration of ionotropes and midazolam infusions, recurrent of fits, complication were noted. They were followed for one month. Then the neurological status at the end of one month was reassessed. Their neurological status was compared with the previous neurological status.

Outcome was determined by the following variables. Complete recovery with no neurological sequel, and neurological sequele Death, AMA, Observations were entered as tables and percentage charts. According to the final outcome the children were divided into two groups. Death were taken as poor outcome group and those children recovered completely with or without any new neurological sequel were taken as good outcome. Predictors of poor outcome were analyzed for the following risk factors compared with good outcome groups and considered statistically significant if p value is <0.05.

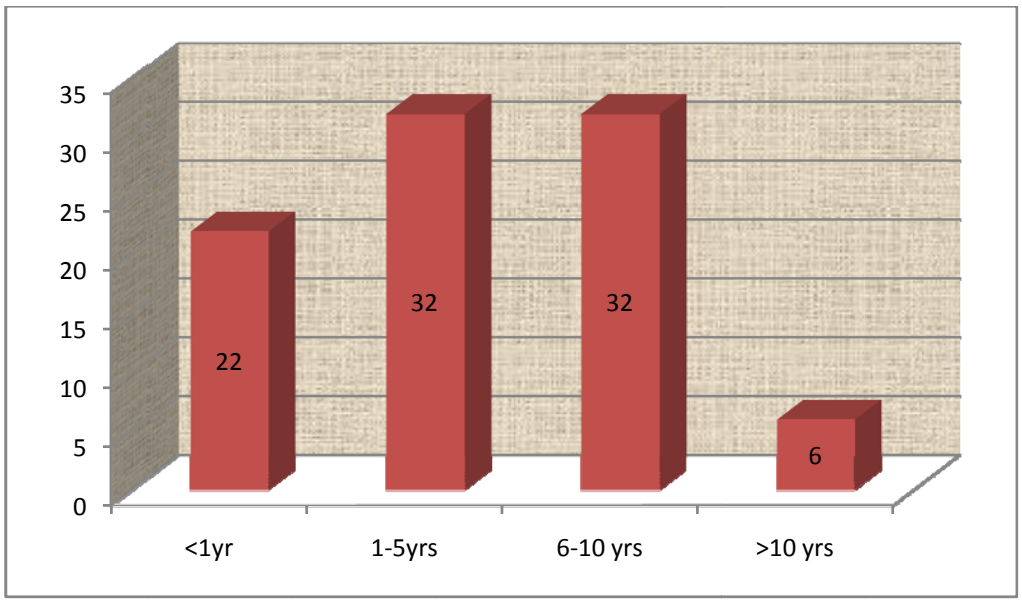
RESULTS AND ANALYSIS

A total of 92 cases of status epilepticus were included in the present study

1.1. AGE DISTRIBUTION

Age in years	No of cases	Percent
< 1	22	23.9
1- 5	32	34.8
6-10	32	34.8
> 10	6	6.5
Total	92	100.0
Mean: 4.9 years Range: 2 months-11 years S.D: 41.241		

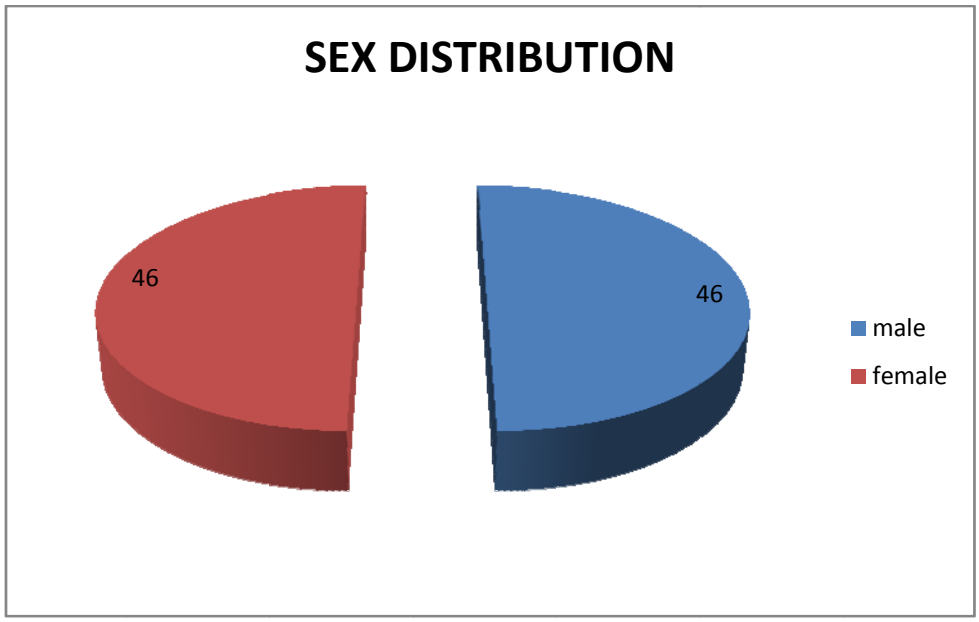
In this study Status epilepticus in children < 1year account for 22 cases 23.9 % of the total cases (n=92) and 54 cases 58.6 % out of the total cases were children < 5 years. Almost 60% of them were in the group < 5 years. No of cases >10yrs was only 6.5%, 6 cases. The mean age of the patient in the present study was 4.9 yrs. The youngest age being 2 month. The maximum age being 11 years.



1.2. SEX DISTRIBUTION

SEX	TOTAL NO OF CASES (n=92)	PERCENTAGE
MALE	46	50.0
FEMALE	46	50.0

Out of 92 children with SE both sex were equal in number. Male children were 46 (50%) and female children were 46 (50%).



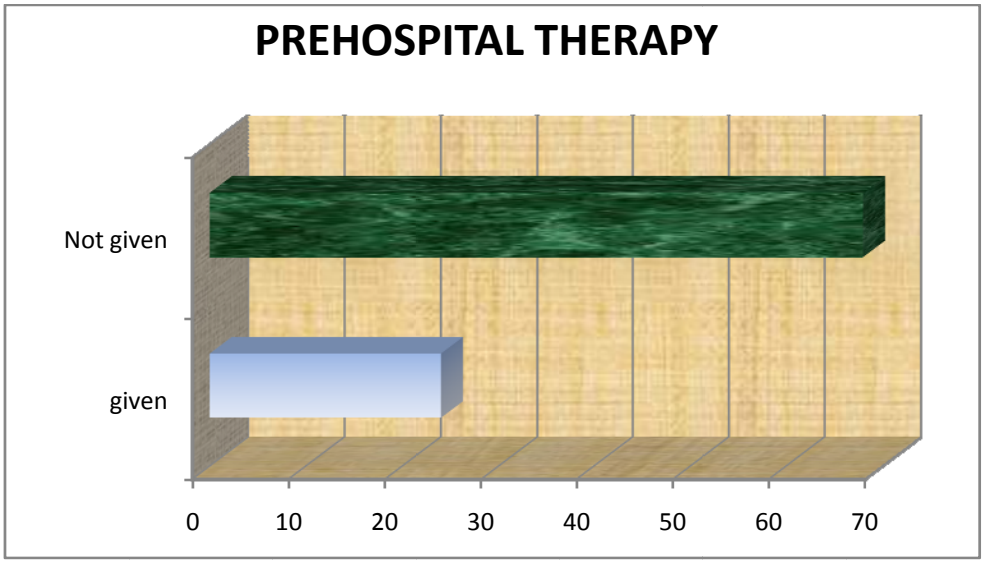
1.3. REFERRAL AND TRANSPORT

Out of 92 cases, 20 cases (21.7 %) were referred from GH. 5 Cases 5.4 % from PHC and 14 cases(15.2%) were referred from private hospital. 53 Cases (57.6%) reached our hospital without treatment or referral.

1.4. PREHOSPITAL THERAPY

Prehospital therapy with AED given to 24 patients 26%, remaining 68 (74%) not received prehospital therapy with AED, though some of them had been referred. Out of 23 children who had received prehospital therapy only 16 had proper prehospital therapy.7 children had received improper medication or drug dosage. 16 children received injection diazepam, 6 children received upto phenytoin and 1 children received phenobarbitone as Prehospital therapy AED.

Prehospital therapy	No of cases	Percentage
Given	24	26.1 %
Not given	68	73.9%
Total	92	100%



1.5.DURATION OF TRANSPORT

Distance travelled	No of cases	Percentage
<5 km	16	17.4
5-10 km	22	23.9
10-20 km	16	17.4
20-40 km	25	27.2
>40 km	13	14.1
Total	92	100
Mean: 19.15 Km Range: 0.5-60 Km S.D: 16.2697		

Of the 92 children 16 cases17.4% of the children with status epilepticus reached TVMCH in less than 5km; 22cases from5-10 km; 16 cases from 10-20 km; 25cases from 20-40 km and 13 cases from more than 40 km. Mean duration being 19 km. Minimum duration travelled was 0.5km and maximum duration was 60 km.

1**.6. DURATION OF SEIZURE AT THE TIME OF ARRIVAL**

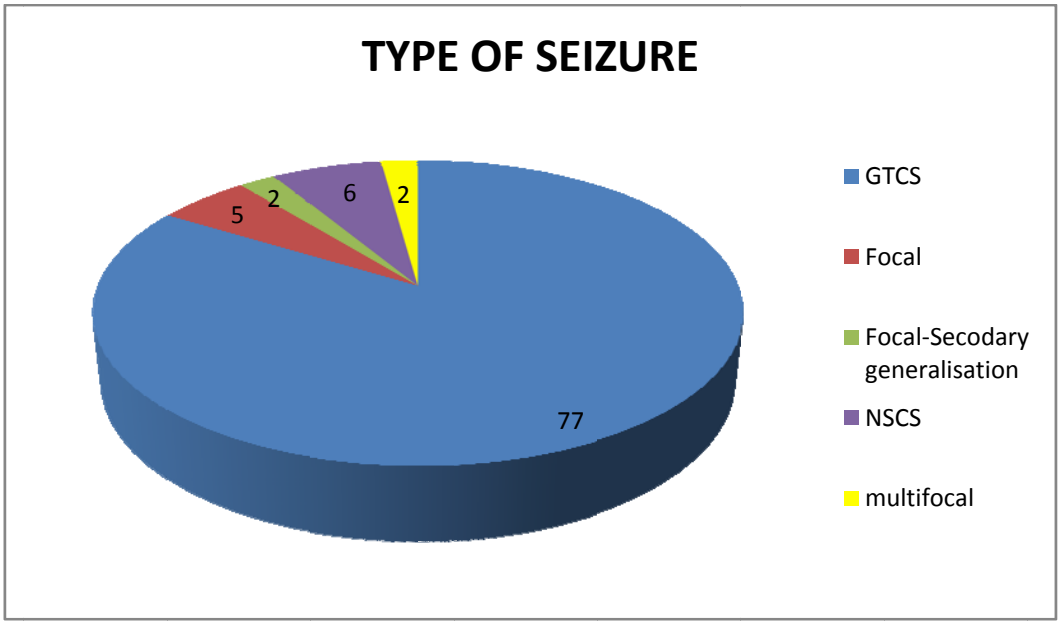
Duration of seizure in min	No of cases n=92	Percentage
30-60 min	75	81.5%
>60 min	17	18.5%
Mean : 78.12 min Range: 30 min-10 hrs SD : 104.938		

81.5% (75) of the cases reached ER within 30-60 mins duration of seizure and 18.5 % (17) reached with seizure more than 60 min. Mean duration of seizure was 78.12 minutes. Minimum duration was 30 mins and maximum duration was 10 hrs.

1.7.SEIZURE TYPE

Type of seizure	No of cases	Percentage
GTCS	77	83.7
FOCAL	05	5.4
FOCAL-S	02	2.2
MULTIFOCAL	02	2.2
NSCE	06	6.5
TOTAL	92	100

CSE accounts about 93.5% of the total seizure during arrival. The commonest among them was GTCS - 77 (83.7%). Focal seizure accounts about 8 % (7 cases). Out of which 2 cases presented with secondary generalisation.Two cases (2.2%) were multifocal. Six cases (6.5 %) were NCSE, of which 4 cases were of initial GTCS and 2 cases were of initial focal seizure.

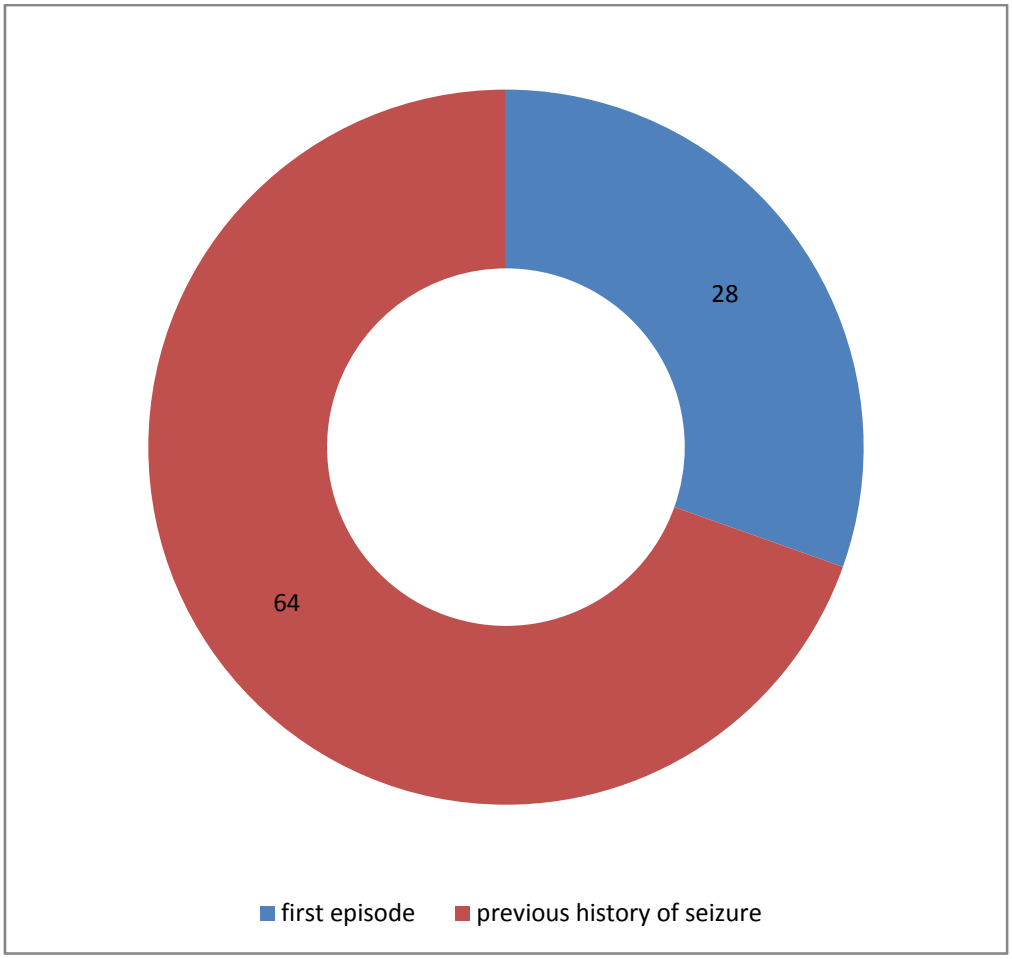


1.8. SEIZURE EPISODE

28 cases 35% presented with first episode of seizure and 64 cases 65% with previous history of seizure.

Seizure episode	No of cases(n=92)	Percentage
First episode of seizure	28	35.0%
Previous episode of seizure	64	65.0%

SEIZURE EPISODE



1.9. FEBRILE OR AFEBRILE SEIZURE

H/O FEVER	No of cases	Percentage
Febrile	65	70.7
Afebrile	27	29.3
Total	92	100

From above table 70.7% (65) cases presented with fever and 29.3% (27) cases were afebrile.

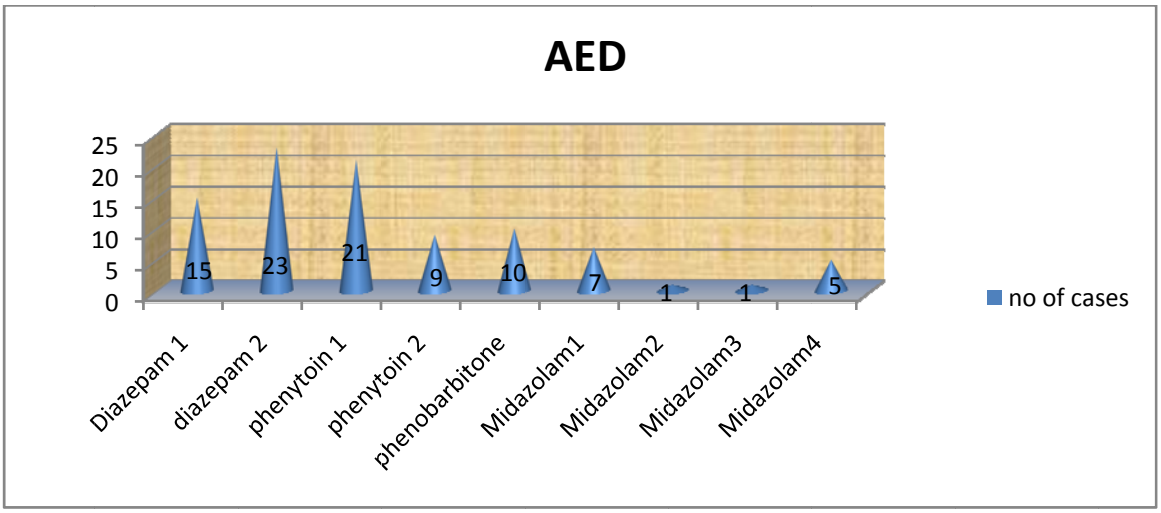
1.10.CO MORBID FACTORS

SI NO	Complications	No of cases (n=92)
1	Intubation on arrival	9 (9.8%)
2	Decompensated shock	8(8.7%)
3	Hypoglycaemia	9((9.8%)
4	Hypocalcaemia	5(5.4%)
5	Acidosis	18 (19.6%)
6	Raised ICT	8(8.7%)

In children presenting with SE at the time of arrival, the most common Co morbid factor is acidosis in 18 cases (19.6%).

1.11. AED TREATMENT RESPONSE

AED	No of cases n=92	Percentage
Diazepam 1 dose	15	16.3
Diazepam 2 dose	23	25.0
Phenytoin 1 dose	21	22.8
Phenytoin 2 dose	09	9.8
Phenobarbitone	10	10.9
Midazolam 1µgm/Kg/min	07	7.6
Midazolam 2 µgm/Kg/min	01	1.1
Midazolam 3 µgm/Kg/min	01	1.1
Midazolam 4 µgm/Kg/min	05	5.4



Out of 92 cases, in 15 cases (16.3%) seizure stopped with 1dose of IV diazepam and 23 cases (25%) responded to 2nd doses of IV diazepam. In 48 cases (41.3%) seizure terminated with 2 doses of diazepam. 30 cases responded to IV Phenytoin. In10 cases seizure terminated with Phenobarbitone IV. Out of 14 cases seizure terminated with Midazolam 1µgm/kg/min in 7 cases, Midazolam 2µgm/kg/min in 1 cases, Midazolam 3µgm/kg/min in 1 cases , Midazolam 4µgm/kg/min in 5 cases.

1.12. REFRACTORY SEIZURE

In this study 15% (14 out of 92) had refractory SE. In the 7 cases survived with RSE, seizure controlled with minimum of midazolam infusion 1μgm/Kg/min to maximum of 4μgm/Kg/min.

1.13 ETIOLOGY

SI NO	Final diagnosis	No of cases(n=92)	Percentage
1	Febrile seizure	17	18.5
2	Acute CNS infection	18	19.6
3	Remote causes	25	27.2
4	Idiopathic SD	15	16.3
5	Neurocutaneous syndrome	02	2.2
6	IE metabolism	02	2.2
7	Non compliance	05	5.4
8	CNS haemorrhage	02	2.2
9	Systemic illness	03	3.3
10	Tumour	03	3.3

From the above table, the most common etiology associated with status epilepticus in 92 children was remote causes 27.2% (25 cases), next being acute CNS infection 19.6% (18 cases), febrile seizure 18.5% (17 cases) and cryptogenic/idiopathic SD 16.3% (15 cases).

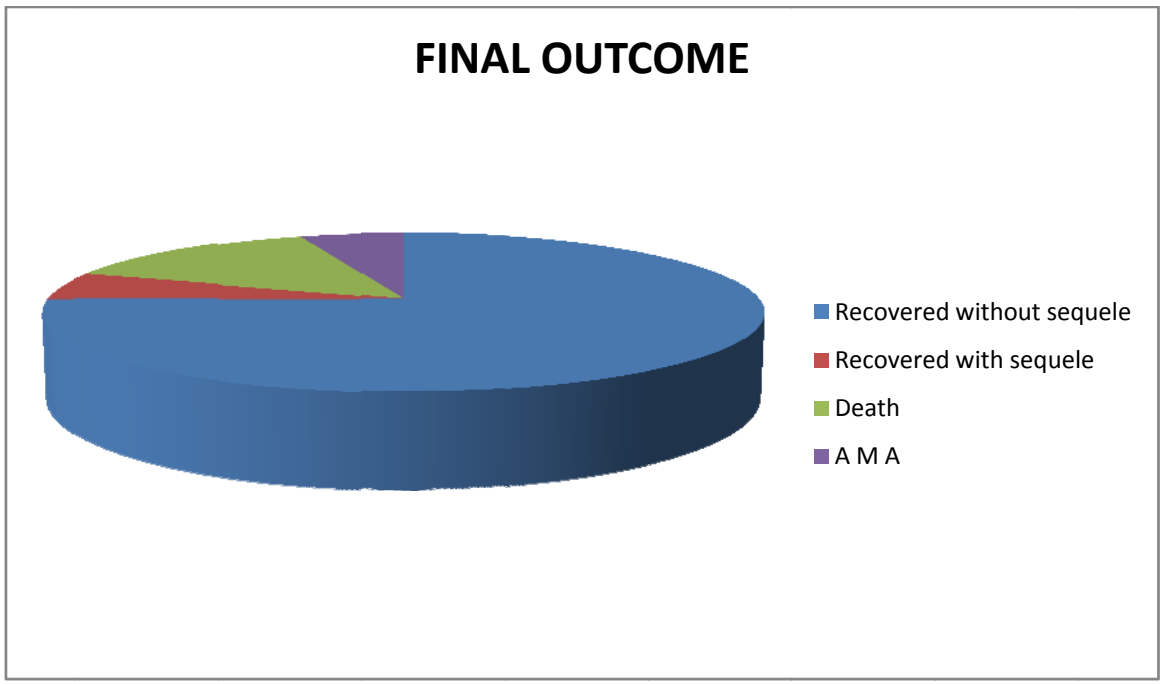
CSF Analysis : CSF analysis was done for 21 children, 6 cases had normal CSF finding.15 cases had abnormal values.

CT brain : done for 44 cases.30 cases had abnormal findings.MRI done for 9 cases,all showed abnormal findings.

1.14. FINAL OUTCOME

Outcome	No of cases n=92	Percentage
Recovered without sequele	69	75.0
Recovered with sequel	05	5.5
Death	13	14.0
AMA	5	5.5
Total	92	100

In this study out of 92 cases, 74 cases (75.0%) recovered. Among those recovered, 5 cases (5.5%) recovered with new neurological sequele; 13 cases (14%) died and 5 cases (5.5%) discharged against medical advice.



2. ANALYSIS OF OUTCOME

Out of 92 cases, 5 went on AMA and didn't complete the study to know the outcome. So the outcome of remaining 87 cases are analysed here.

2.1 AGE AND OUTCOME

Age	Recovered	Death	Total
<1yr	19 (86.4%)	3 (13.6%)	22 (100%)
1-5yrs	27 (87.1%)	4 (12.9%)	31 (100 %)
6-10yrs	25 (83.3%)	5 (16.7%)	30 (100%)
>10yrs	3 (75.0%)	1 (25.0%)	4 (100%)
Total	74 (85.1%)	13 (14.9%)	87 (100%)

Chi^2: 0.519 df: 3 p- Value: **0.915**

The above table explains the outcome across the various age groups. Of the total 87 patients 74 (85.1%) have recovered and 13 (14.9%) have died. Almost the same proportion is maintained across the all age groups. So the age is not having significant association with the outcome (p- Value > 0.05).

2.2. DURATION OF TRAVEL AND OUTCOME

Distance KM	Recovered	Death	Total
<5	15(93.8%)	1(6.3 %)	16(100%)
5-10	18(100%)	0(0 %)	18(100 %)
11-20	13(81.3 %)	3(18.8 %)	16(100 %)
21-40	22(88%)	3(12%)	25(100 %)
>40	6 (50 %)	6 (50%)	12(100 %)
Total	74(85.1 %)	13(14.9 %)	87(100%)

Chi^2: 16.070 df: 4 p- Value: **0.003**

The above table explains the relationship between distance of travel and the outcome. Outcome is good among cases travelled less than 10 km and mortality is very low among children travelled less than 10 km. Mortality is very high among 12 children travelled more than 40 km. Out of 12 cases, 6 cases (50%) recovered and 6 cases (50%) died. It shows that lower the distance better the outcome and higher the distance higher the bad outcome (death). The association is also statistically significant (p –Value < 0.05).

2.3. PREHOSPITAL THERAPY

Prehospital therapy	Recovered	Death	Percentage
Yes	17 (73.9 %)	6 (26.1 %)	64 (100%)
No	57 (89.1%)	7 (10.9 %)	23 (100%)
Total	74 (85.1%)	13 (14.9%)	87 (100%)

Chi^2: 3.055 df: 1 p- Value: **0.08**

The above table shows that the cases had prehospital therapy had poor outcome (26.1%) than the case who didn't receive prehospital therapy (7%). But this difference is not statistically significant (p- value > 0.05).

2.4. DURATION OF SEIZURE BEFORE ARRIVAL

Duration of seizure Minutes	Recovered	Death	Total
30-60	66(93.0%)	5(7.0 %)	71(100%)
>60	8(50.0 %)	8(50.0 %)	16(100%)
Total	74(85.1 %)	13(14.9 %)	87(100%)

Chi^2: 18.959 df: 1 p- Value: **0.000**

The above table shows that prolonged duration of the seizure before arrival to ER had poor outcome. This is also statistically significant (p- value <0.05).

2.5 SEIZURE TYPE AND OUTCOME

Fit Type	Recovered	Death	Total
GTCS	64(86.5%)	10(13.5%)	74(100%)
NCSE	3(50%)	3(50%)	6(100%)
FOCAL	0(0%)	5(100%)	5(100%)
FOCAL-S	0(0%)	2(100%)	2(100%)

Chi^2: 7.151 df: 3 p- Value: **0.067**

Among the seizure type death occurred in children with GTCS and NCSE. Out of 74 cases with GTCS 10 cases died and in NCSE 3 cases(50%) died out of 6 cases(100%).This is statistically not significant. (p- Value:>0.05).

2.6. FEVER ASSOCIATION AND OUTCOME

Fever H/O	Recovered	Death	Total
Febrile	54 (84.4%)	10 (15.6%)	64 (100%)
Afebrile	20 (87%)	3 (13%)	23 (100%)
Total	74 (85.1%)	13 (14.9%)	87 (100%)

Chi^2: .089 df: 1 p- Value: **0.766**

From the above table all the proportions of outcome are maintained almost equally in both the febrile and afebrile group. The difference is not statistically significant (p value > 0.05).

2.7. NEUROLOGICAL STATUS

Neurological status	Recovered	Death	Total
Normal	48 (90.6%)	5(9.4%)	53(100%)
Abnormal	26(76.5%)	8(23.5%)	34(100%)
Total	74(85.1%)	13(14.9%)	87(100%)

Chi^2: 3.238 df: 1 p- Value: **0.072**

Among the previously neurological abnormal children of 34 cases, 23.5% (8) cases had poor outcome. Only 9.4% (5) of the neurologically normal children (n=53) had poor outcome. But this different is not statistically significant (p- value >0.05).

2.8. COMORBID FACTORS

SI NO	Risk factors	Recovered (n=74)	Death (n=13)	Total (n=87)	p value
1	Shock	6(8.11%)	2(15.38%)	8(9.2%)	**0.000**
2	Fever	54(72.97%)	10(76.92%)	64(73.56%)	0.766
3	Raised ICT	6 (8.10%)	0 (0%)	6(6.897%)	0.287
4	Intubation	11(14.86%)	8(69.54%)	19(21.84%)	**0.000**
5	SaO2 on arrival(low)	30(40.54%)	9(69.23%)	39(44.83%)	**0.004**
6	Hypoglycemia	5(6.76%)	4(30.76%)	9(10.34%)	**0.015**
7	Hypocalcemia	8(10.81%)	8(69.54%)	16(18.39%)	1.000
8	Acidosis	8(10.81%)	8(69.54%)	16(18.39%)	**0.000**
9	Delay in regain consciousness after seizure	22(29.73%)	2(15.38%)	24(27.58%)	0.942

From the above table the risk factors significantly affecting the outcome are hypoxia at the time of arrival, decompensated shock, respiratory failure requiring intubation, hypoglycemia and acidosis. (p- value <**0.05**).

2.9 AED TREATMENT RESPONSE AND OUTCOME

AED Treated	Recovered	Death	Total
Diazepam 1	14(93.3%)	1(6.7%)	15(100%)
Diazepam 2	22(95.7%)	1(4.3%)	23(100%)
Phenytoin 1	18(85.7%)	3(14.3%)	21(100%)
Phenytoin 2	7(100%)	0(0%)	7(100%)
Phenobarbitone	6(66.7%)	3(33.3%)	9(100%)
Midazolam 1µgm/Kg/min	4(80%)	1(20%)	5(100%)
Midazolam 2µgm/Kg/min	1(100%)	0(0%)	1(100%)
Midazolam 3µgm/Kg/min	0(0%)	1(100%)	1(100%)
Midazolam 4µgm/Kg/min	2(40%)	3(60%)	5(100%)
Total	74(85.1%)	13(14.9%)	87(100%)

Chi^2: 20.427 df: 8 p- Value: **0.009**

From the above table if the seizure control is poor with initial first line AED, mortality is very high.In children receiving Midazolam infusion (3-4µgm/Kg/min) had poor outcome.This is also statistically significant p- Value < 0.05.

2.10. ETIOLOGY AND OUTCOME

SI NO	Final diagnosis	Recovered	Death	Total Percentage
1	Febrile seizure	17 (100%)	0(0%)	17(100%)
2	Acute CNS infection	13(76.5%)	4(23.5%)	17(100%)
3	Remote causes	19(76%)	6(24%)	25(100%)
4	Idiopathic SD	12(92.3%)	1(7.7%)	13(100%)
5	Neurocutaneous syndrome	02(100%)	0(0%)	2(100%)
6	IE metabolism	01(50%)	01(50%)	2(100%)
7	Non compliance	04(80%)	01(20%)	5(100%)
8	CNS haemorrhage	02(100%)	0(0%)	2(100%)
9	Systemic illness	03(100%)	0(0%)	3(100%)
10	Tumour	01(100%)	0(0%)	1(100%)
TOTAL		74(85.1%)	13(14.9%)	87(100%)

In this study 87 children out of 92 cases who completed the study, 74 cases recovered and 13 cases died due to varied etiology. All the febrile seizure cases presented as SE had good outcome. Acute CNS infection, remote causes, non compliance of AED, IEM, are the etiological factors influence the poor outcome.

2.11. RECOVERED WITH SEQUELE AND OUTCOME

SI. No	Etiology	Total cases	Recovered with sequele
1	Acute CNS infection	18	2 (11.11%)
2	CNS haemorrhage	2	1 (50%)
3	Idiopathic SD	15	1 (6.66%)
4	Remote cause	25	1 (4.0%)
Total			5 cases

Among 87 cases, 5 cases developed neurological sequele. The most common etiological agents associated with sequele are acute CNS infection, CNS haemorrhage, Idiopathic SD, remote cause.

DISCUSSION

In this study there were total of 92 cases of status epilepticus within the study period. The mean age of the patient in the present study was 4.9yrs.The youngest age being 2 month and the eldest being 11 years. Children < 1year account for 22 cases 23.9 % of the total cases (n=92) and 54 cases 58.6 % out of the total cases were children < 5 years. Almost 60% of them were in the group < 5 years. No of cases >10yrs was only 6.5%, 6 cases. Out of 92 children, male children were 46 (50%) and female children were 46 (50%). **Sachin Admuthe, et al**,[53] in his study showed mean age was 4.5yrs and weak evidence of difference of sex.

Study	Mean age
Sachin Admuthe et al	4.5yrs
Maytal J et al [54]	5 yrs
R K Sing's et al[55]	3.4yrs

Out of 92 cases, 20 cases (21.7 %) were referred from GH. 5 Cases (5.4 %) from PHC and 14 cases (15.2%) were referred from private hospital. 53 Cases (57.6%) reached our hospital without any treatment or referral. Of the 92 children, 16 cases17.4% of the children with status epilepticus reached TVMCH in less than 5km; 22 cases from 5-10 km; 16 cases from 10-20 km; 25cases from 20-40 km and 13 cases from more than 40 km. Mean duration being 19 km. Minimum duration travelled was 0.5 km and maximum duration was 60 km.

Prehospital therapy with AED given to 24 patients (26.1%), remaining 68 (73.9%) didn't receive any prehospital therapy with AED, though some of them had been referred. Out of 23 children who had received prehospital therapy only 16 had proper prehospital therapy.7 children had received improper medication or drug dosage. 16 chilldren received

injection diazepam, 6 children received upto phenytoin and 1 children received phenobarbitone as Prehospital therapy AED.

81.5 % (75) of the cases reached ER within 30-60 min and 18.5 % (17) reached with seizure more than 60 min. Mean duration of seizure was 78.12 minutes. Minimum duration of seizure was 30mins and maximum duration was 10 hrs. **K. Eriksson et al**,[45] study concluded that the association between treatment delay and response became significant after 30 minutes when this was analyzed as a single variable (p = 0.003)

CSE accounts about 93.5% of the total seizure during arrival. The commonest among them was GTCS - 77 (83.7%). Focal seizure accounts about 8 % (7 cases). Out of which 2 cases presented with secondary generalisation. Two cases (2.2%) were multifocal. Six cases (7 %) were NCSE, of which 4 cases were of initial GTCS and 2 cases were of initial focal seizure.

28 cases 35% presented with first episode of seizure and n=51 65% with previous history of seizure. Out of 92 cases, 70.7% (65) cases presented with fever and 29.3% (27) cases were afebrile. **Garzon et al**[56] study showed 40.6% with no prior history of seizure.43% in **Mah JK et al**,[57] **Kalra veena et al** [58]study showed53.3% with first episode of seizure.

In this study risk factors associated with status epilepticus are Intubation on arrival, decompensated shock, hypoglycaemia, acidosis, raised ICT. The above risk factors significantly affect the outcome of SE in children. Among them acidosis is the most common risk factor – 18 (19.6%).

Out of 92 cases, in 15 cases (16.3%) seizure stopped with 1dose of IV diazepam and 23 cases (25%) responded to 2nd doses of IV diazepam. In 48 cases (41.3%) seizure terminated with 2 doses of diazepam. 30 cases responded to IV Phenytoin. In10 cases seizure terminated with Phenobarbitone IV. Out of 14 cases seizure terminated with Midazolam 1µgm/kg/min in 7 cases, Midazolam 2µgm/kg/min in 1 cases, Midazolam 3µgm/kg/min in 1 cases, Midazolam 4µgm/kg/min in 5 cases.

In this study 15% (14 out of 92) had refractory SE and out of them, 7 survived and 5 died. In the 7 cases survived with RSE, seizure controlled with minimum of Midazolam infusion 1µgm/Kg/min to maximum of 4µgm/Kg/min. **Garson et al**,[56] study showed 11.3% incidence of RSE. In this study cases gone for RSE had poor outcome.

The most common aetiology associated with status epilepticus in 92 children was remote causes 27.2% (25 cases), next being acute CNS infection 19.6% (18 cases), febrile seizure 18.5% (17 cases) and cryptogenic/idiopathic SD 16.3% (15 cases).**Hui Ac et al, study**[59] shows that acute CNS infection was a predictor of poor outcome. **Murthy JM et al**[60] study shows CNS infection accounts for significant number of cases.

In this study out of 92 cases, 74 cases (75.0%) recovered. Among those recovered, 5 cases (5.5%) recovered with new neurological sequele; 13 cases (14%) died and 5 cases (5.5%) discharged against medical advice.

Discussion onAnalysis of Outcome

Out of 92 cases, 5 went on AMA and didn't complete the study to know the outcome. So tsshe outcome of only 87 cases are analysed here.

Table 2.1 explains the outcome across the various age groups. Of the total 87 patients 74 (85.1%) have recovered and 13 (14.9%) have died. Almost the same proportion is maintained across the all age groups. So the age is not having significant association with the outcome (p- Value > 0.05).

Table 2.2 explains the relationship between distance of travel and the outcome. Outcome is good among cases travelled less than 10 km and mortality is very low among children travelled less than 10 km. Mortality is very high among 12 children travelled more than 40 km. Out of 12 cases, 6 cases (50%) recovered and 6 cases (50%) died. It shows that lower the distance better the outcome and higher the distance higher bad outcome (death). The association is also statistically significant (p –Value < 0.05). . **K. Eriksson, et al,**[45] study

concluded that the association between treatment delay and response became significant after 30 minutes when this was analyzed as a single variable (p = 0.003).

The above table 2.3 shows that the cases had prehospital therapy had poor outcome 6 cases, (26.1%) than the case who didn't receive prehospital therapy 7 cases(10.9%). But this difference is not statistically significant (p- value > 0.05). Those cases who received prehospital therapy had poor outcome (26.1%). This may be due the improper drug dosage at lower level hospitals and delayed referral. On contrary those who didn't have prehospital therapy had come straight to the tertiary care hospital where appropriate management protocol is followed. **Alldredge BK, et al**[61] data suggest that prehospital administration of diazepam may shorten the duration of SE in children and simplify the subsequent management of these patients in the emergency department. **KL Kwong,et al**[62],also supports that BZD premedication reduces adverse outcome

Table 2.4 shows that prolonged duration of the seizure before arrival to ER had poor outcome. This is also statistically significant (p- value <0.05). **Gulati S,et al**[36] ,**Karla Veena et al,**[58] also in their study found that seizure duration more than 45 minutes is significantly associated with higher mortality. **KL Kwong,et al** [62] study also supports that seizure duration more than 60 minutes had adverse outcome. **Towne AR et al**,[51] the group with SE lasting < 1 hour had a lower mortality as compared with seizure duration more than or equal to1 hour.

From the table 2.6 it is observed that all the proportions of outcome are maintained almost equally in both the febrile and afebrile group. The difference is not statistically significant (p value > 0.005).

Among the seizure type, death occurred in children with GTCS and NCSE. Out of 74 cases with GTCS 10 cases died and in NCSE 3 cases(50%) died out of 6 cases(100%).This shows that number of death among children with GTCS is common but in NCSE 3 cases(50%) died out of 6 cases(100%). Other studies also shows the same results.

If the seizure control is poor with initial first line AED, mortality is very high.In children receiving Midazolam infusion (3-4µgm/Kg/min) had poor outcome.

Among the previously neurological abnormal children of 34 cases, 23.5% (8) cases had poor outcome. Only 9.4% (5) of the neurologically normal children (n=53) had poor outcome. But this different is not statistically significant (p- value >0.05). **KL Kwong,et al**[62] in his study observed Paediatric patients with status epilepticus who had normal neurodevelopmental status before the onset of an attack and who did not sustain an acute insult to the central nervous system or have progressive encephalopathy had good prognosis. From the table 2.8 the risk factors significantly affecting the outcome are hypoxia at the time of arrival, decompensated shock, respiratory failure requiring intubation and acidosis. (p-value <0.05). **Kalra Veena et al**,[58] study showed presence of septic shock (p-0.001) were associated with significant mortality.

FINAL OUTCOME

Out of 92 case, 87 cases who completed the study, 74 cases recovered and 13 cases died due to varied etiology. All the febrile seizure cases presented as SE had good outcome. Acute CNS infection, remote causes, non compliance of AED, IEM, are the etiological factors influence the poor outcome. **KL Kwong et al,**[62] study support the influence of etiological agents like symptomatic etiology, remote cause, refractory SE on the poor outcome. **B G R Neville,et al**,[422] study conclude that febrile CSE is the commonest single group with a good prognosis in sharp distinction to CSE related to central nervous system infections which have a high mortality.

Among 74 cases survived, 5 cases(6.7%) developed neurological sequele.3cases had GTCS, one had NCSE, and another one case had Focal to secondary generalisation.3 cases were less than 1year and 2 cases were more than 10 years among chidren developed sequele(Aphasia-2,Hemiparesis-1,Cognitive dysfunction-2). Out of 5 cases 4cases associated

with acidosis, one case associated with shock.Acute CNS infection (2 cases), Remote cause (2 cases) and AED Drug non compliance (1 case). **Maytal J, et al**[54] study shows that new neurologic deficits were found in 17 (9.1%) of the 186 survivors. All of the deaths and 15 of the 17 sequele occurred in the 56 children with acute or progressive neurologic insults. **Dunn W**[63] study 18 out of 114 cases developed neurological deficit and 11% in **Simon J et al**[64] study.

CONCLUSION

1. Children with status epilepticus who had normal neurodevelopment status before the onset of an attack and who did not sustain an acute insult to the central nervous system, had favourable outcomes.
2. The local population has restricted access to medical facilities and treatment before admission to our hospital is often inadequate. Seizures therefore last longer before medical care is reached, and are thus more refractory to treatment. This factor might be more important. Duration and distance travelled to seek medical advice are also important risk factors that influencing the poor outcome.

   a) Hence early institution of proper time framed therapy even by the nearest hospital will improve the outcome.Atleast proper prehospital therapy like the use of IM/IV midazolam or IV/ Rectal diazepam will have good outcome.

   b) Airway stabilization with supplementary oxygen while transporting the child from periphery could result in better outcome in status epilepticus.
3. Although the outcome is dependent on aetiology, appropriate early management along with specific AED therapy as per protocol may reduce some of the morbidity associated with CSE.
4. Acute CNS infection is one of the independent risk factor for poor outcome .Hence appropriate empirical antimicrobials to be given in a suspected case of acute CNS examination. All children should be routinely immunised for Hib, pneumococcal vaccines.
5. Individual risk factors and complication to be anticipated and early intervention for complication like shock respiratory failure, aspiration, hypoglycemia or hyperglycemia, dyselectrolytemias to achieve good outcome.

6. Proper prehospital therapy, early referral, proper care while transporting, anticipating risk factors involved, and protocol based approach uniformly at all hospital can reduce the mortality due to status epilepticus in children.

SUMMARY

- In this study Status epilepticus in children < 1year account for 22 cases 23.9 % of the total cases (n=92) and 54 cases 58.6 % out of the total cases were children < 5 years. Almost 60% of them were in the group < 5 years.
- The mean age of the patient in the present study was 4.9 yrs.The youngest age being 2 month.
- Out of 92 children with SE both sex were equal in number.
- Of the total 87 patients who completed the study 74 (85.1%) have recovered and 13 (14.9%) have died.
- Outcome is good among cases travelled less than 10 km and mortality is very low among children travelled less than 10 km. Mortality is very high among 12 children travelled more than 40 km. Out of 12 cases, 6 cases (50%) recovered and 6 cases (50%) died.
- Those cases who had prehospital therapy had poor outcome (26.1%) than the case who didn't receive prehospital therapy (7%). But this difference is not statistically
- Prolonged duration of the seizure before arrival to Emergency Room had poor outcome.
- Among the seizure type death occurred in children with GTCS and NCSE. Out of 74 cases with GTCS 10 cases died and in NCSE 3 cases(50%) died out of 6 cases(100%).
- If the seizure control is poor with initial first line AED, mortality is very high.In children receiving Midazolam infusion (3-4µgm/Kg/min) had poor outcome.
- All the proportions of outcome are maintained almost equally in both the febrile and afebrile group.

- Among the previously neurological abnormal children of 34 cases, 23.5% (8) cases had poor outcome. Only 9.4% (5) of the neurologically normal children (n=53) had poor outcome
- The risk factors significantly affecting the outcome are hypoxia at the time of arrival, decompensated shock, respiratory failure requiring intubation, hypoglycaemia and acidosis
- 87 children out of 92 cases who completed the study, 74 cases recovered and 13 cases died due to varied etiology. All the febrile seizure cases presented as SE had good if the seizure control is poor with initial first line AED, mortality is very high.In children receiving Midazolam infusion (3-4µgm/Kg/min) had poor outcome. outcome. Acute CNS infection, remote causes, non compliance of AED, IEM, are the etiological factors influence the poor outcome.
- Among 87 cases, 5 cases developed neurological sequele.The most common etiological agents associated with sequele are acute CNS infection, Intra Cerebral haemorrhage, Idiopathic SD, remote cause.

ANNEXURE PROFOMA

Serial No :

Hospital No :

Ward Admitted :

Name :

Age :

Sex :M/F

Address

Contact No :

D. O. A

D. O. D

Time of arrival

Referred From

1.Nil

2.PHC

3.GH

4. Private Hospital

5.OPD

Distance from,TVMCH

1. <5km

2.5-10km

3.10-20km

4.21-40km

5.>40km

Mode of transport

1.hand

2.car

3.auto

4.twowheeler

5.ambulance

6.others

Duration of seizures prior to reaching TVMCHin minutes

Type of seizures

1. GTCS

2. Non convulsive

3. Secondary generalisation

4.others

Number of attacks

Duration :

Risk factor:sleep deprivation/stress/trauma/toxindrugs/

Unprovoked/medical illness/others

Level of sensorium in between attacks. ALOC/normal

H /o ALOC before fits yes/no

H/o head trauma:yes/no

H/o toxin/drug ingestion:yes/no

pre hospital therapy

Drug and route of administration

1.diazepam

2.lorazepem

3.phenytoin/phenobarbitone/midazolam.

4.others.

Past history

h/o status epilepticus yes/no

1.febrile/afebrile

2.date of last episode.

If yes date of occurance

Duration

On AED:

Regular or not

Dosage and duration

Compliance:

Last dose

Development h/o normal delayed

Neurological status before SE: normal /abnormal

If abnormal specify

Co-morbid condition

1

2

3

4

Clinical examination

Appearance : A/V/P/U

Breathing : required BVM: If yes/duration

Required intubation : yes/no how long

SPO2 on arrival

Circulation shock yes or no

If yes: compensated/decompensated

BP on arrival: mmHg

Required fluids/required ionotropes / uncorrected

Pupil

Blood sugar

Management of seizures

25% dextrose/lorazepam/BZD/phenytoin/phenobaritonrefractory seizure

IN PICU

Midazolam /thiopentone/others

Rate of infusion

shock:yes/no

Time taken for full recovery of consciousness: mins /hours

Complication

1.Acidosis

2.pneumonia

3.respiratort failure

4.ICP

5.Shock

6.Hyperthermia

7. neuroligal deficit

8.Hypoglycaemia

9.Hypocalcaemia

Investigation

Hb%

Tc dc pcv

Platelet /peripheral smear study sugar urea creatinine electrolyte LFT

Blood culture

Urine Metabolic Screening

CXR

CSF anlaysis

USG cranium

CT brain / MRI

EEG

Final diagnosis

Outcome

1. Recovered a) with or without sequele

2.AMA

3.Death

ABBREVIATION

SE: STATUS EPILEPTICUS

CSE: CONVULSIVE STATUS EPILEPTICUS

RSE: REFRACTORY STATUS EPILEPTICUS

NCSE: NON CONVULSIVE STATUS EPILEPTICUS

ASE: ABSENT STATUS EPILEPTICUS

PCSE: PARTIAL COMPLEX STATUS EPILEPTICUS

IPPV: INTERMITTENT POSITIVE PRESSURE VENTILATION

CNS: CENTRAL NERVOUS SYSTEM

AED: ANTI EPILEPTIC DRUGS

ACD: ANTI CONVULSANT DRUGS

GABA: GAMMA AMINO BUTERIC ACID

EEG: ELECTRO ENCEPHALOGRAM

NMDA: N-METHYL D-ASPARTATE

GTCS: GENERALISED TONIC CLONIC SEIZURE

CVA: CEREBRO VASCULAR ACCIDENT

GCSE: GENERALISED COVULSIVE SE

BZD: BENZODIAZEPINES

LP: LUMBAR PUNCTURE

PE : PHENYTOIN EQUIVALENT

IV : INTRAVENOUS

CXR : CHEST X-RAY

CT: COMPUTED TOPOGRAPHY

USG : ULTRA SONOGRAPHY

ECG : ELECTRO CARDIOGRAPHY

D.O.A. : DATE OF ADMISSION

D.O.D. : DATE OF DISCHARGE

TVMCH : TIRUNELVELI MEDICAL COLLEGE HOSPITAL

ALOC : ALTERED LEVEL OF CONSCIOUSNESS

IEM : INBORN ERROR OF METABOLISM

ICP : INTRA CRANIAL PRESSURE

PICU : PEDIATRIC INTENSIVE CARE UNIT

A/V / P/U : A – ALERT, V – VERBAL, P – PAIN RESPONSIVE, U – UNCONSCIOUS

BVM : BAG AND MASK VENTILATION

BP : BLOOD PRESSURE

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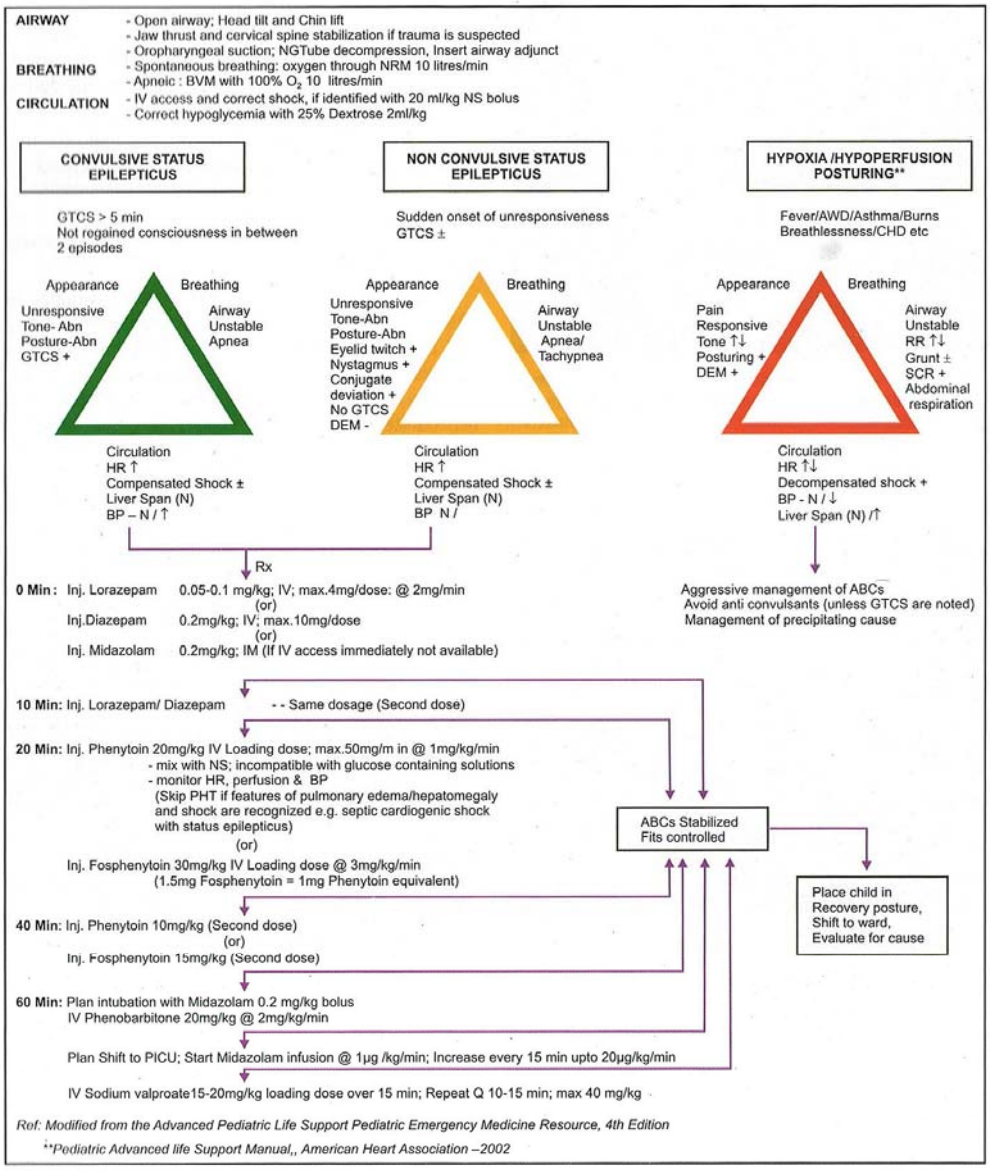
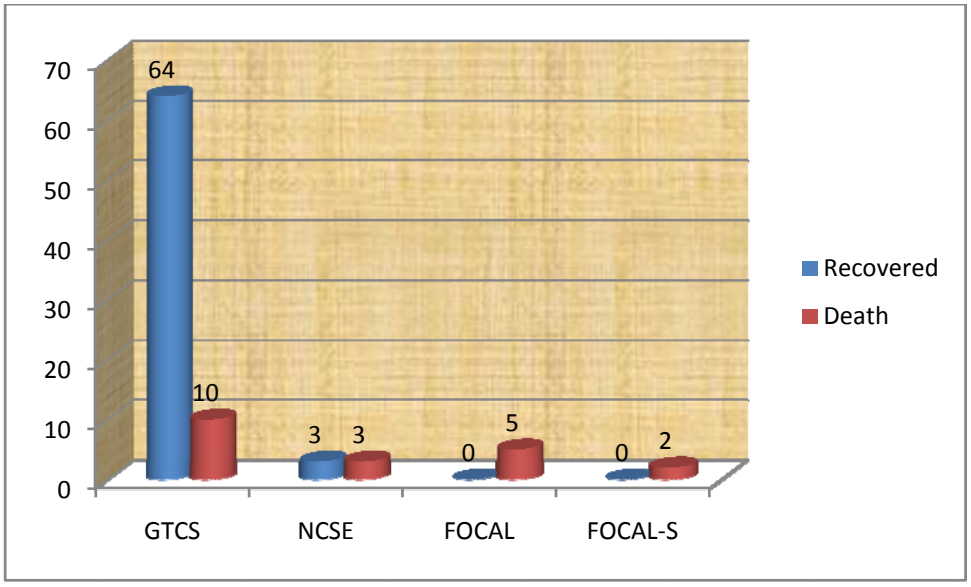
Protocol : Recognition and management of pediatric convulsive and non-convulsive status epilepticus in the emergency department



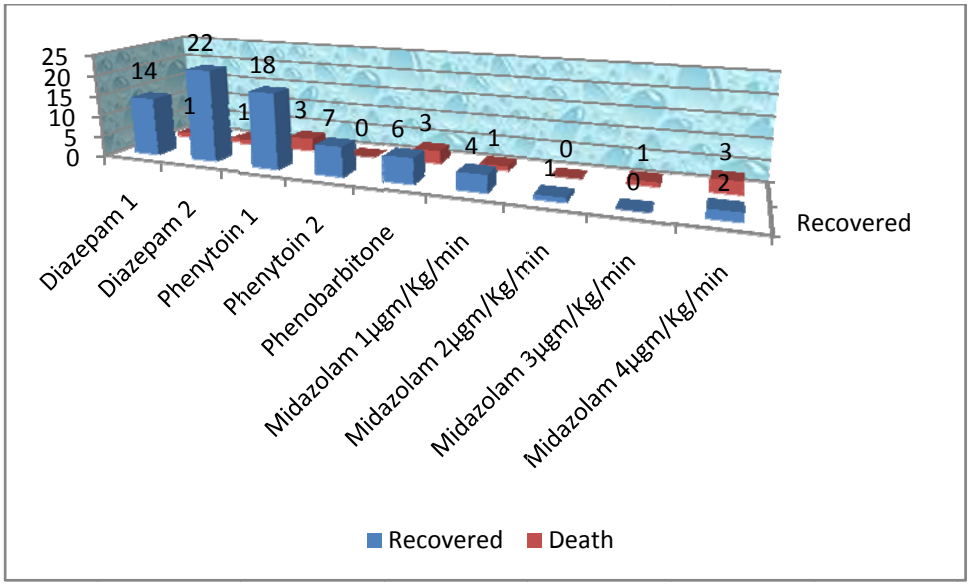
Ref: Modified from the Advanced Pediatric Life Support Pediatric Emergency Medicine Resource, 4th Edition

***Pediatric Advanced life Support Manual,, American Heart Association –2002*

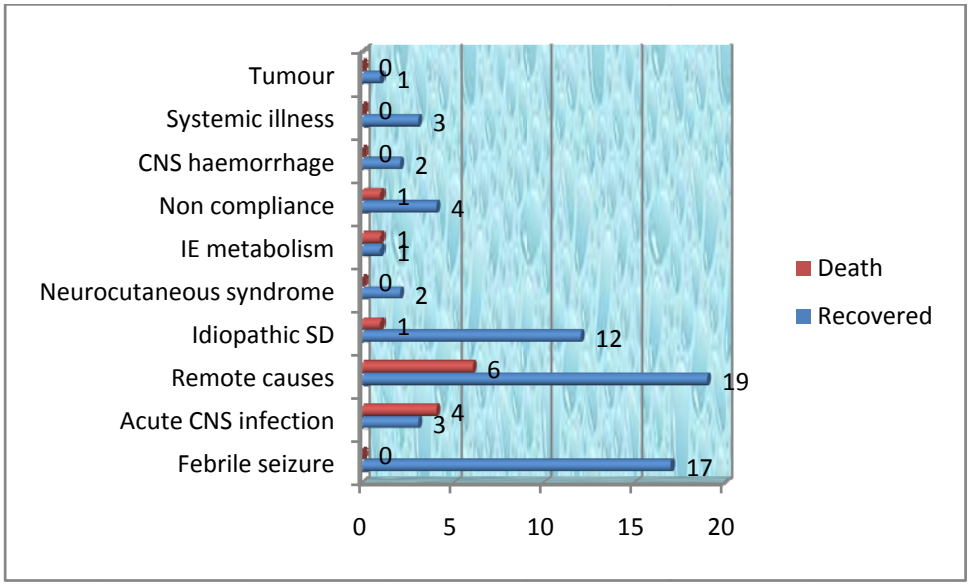
SEIZURE TYPE AND OUTCOME



AED TREATMENT RESPONSE AND OUTCOME



ETIOLOGY AND OUTCOME



MASTER CHART

S.NO	Name	AGE	AGE.GROUP	SEX	REFFROM	DISTANCE	DURAFIT	FITTYPE	FEBAFEB	RISKFACT	neurol.nor.abnor	TRANSP	PREHOSP	DRUG	PASTHO	BVM	INTUBATI	SAO2	SHOCK	GLUCOSE	CALCIUM	AED	ACIDOSIS	ICP	deficit	LP	CTBRAIN	MRI	FINALDIAGNOSIS	outcome
1	Abi	11	< 1	f	self	0.5	30	GTCS	febrile	cns inf	2	bus	nil	nil	n	yes	no	94	no	no	no	2. D2	NO	NO	NO	a	n	-	ACUTE CNS INFECTION	RECOVERED
2	Isaki	27	1- 5	m	self	9.0	30	GTCS	afebrile	cp	1	auto	nil	nil	y	yes	no	92	no	no	no	3. P1	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
3	Durgaraj	24	1- 5	m	self	8.0	40	GTCS	febrile	cns inf	2	auto	nil	nil	n	yes	no	95	no	no	no	4. P2	NO	NO	NO	a	n	-	ACUTE CNS INFECTION	RECOVERED
4	Chellamal	60	1- 5	f	phc	55.0	180	GTCS	febrile	cn/res fail/shoc	2	amb	nil	nil	n	yes	y	85	y	l	no	5. PH	YES	NO	NO	a	n	-	ACUTE CNS INFECTION	EXPIRED
5	Mukesh	12	< 1	m	self	0.8	30	GTCS	febrile	fever	2	car	nil	nil	n	yes	no	94	no	no	no	2. D2	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
6	Uma	84	6-10	f	pvt	0.5	30	FOCAL	febrile	fever	2	bike	nil	nil	n	yes	no	97	no	no	no	2. D2	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
7	Yoga preethika	8	< 1	f	pvt	20.0	60	GTCS	febrile	res fail/shoc	1	amb	yes	diazepam	y	yes	y	88	y	l	no	3. P1	YES	NO	NO	n	n	a	I E METABOLISM	EXPIRED
8	krishnaveni	24	1- 5	f	gh	50.0	240	GTCS	febrile	add	2	amb	yes	diazepam	n	yes	y	93	no	no	no	5. PH	NO	NO	NO	n	n	-	ACUTE CNS INFECTION	AMA
9	Md Basim	108	6-10	m	pvt	13.0	72	GTCS	febrile	cns inf	2	amb	yes	phenytoin	n	yes	no	94	no	h	no	4. P2	YES	YES	YES	a	n	a	ACUTE CNS INFECTION	RECOVERED-S
10	Uma	84	6-10	f	self	7.0	35	MULTI	afebrile	NO RISK	2	bike	nil	nil	n	yes	no	96	no	no	no	4. P2	NO	NO	NO	-	-	-	IDIOPATHIC S D	AMA
11	Monisha	8	< 1	f	pvt	25.0	360	NCSE	febrile	add	1	car	yes	diazepam	n	yes	y	85	no	no	no	9. M4	YES	NO	NO	-	n	-	REMOTE CAUSE	EXPIRED
12	Ranganathan	2	< 1	m	self	60.0	600	NCSE	febrile	cns inf	2	amb	yes	phenytoin	n	yes	y	84	no	no	no	9. M4	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	EXPIRED
13	Mareshwari	36	1- 5	f	pvt	50.0	180	GTCS	febrile	cns inf	2	amb	yes	diazepam	n	yes	y	87	y	no	no	5. PH	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	EXPIRED
14	Mahalakshmi	8	< 1	f	self	7.0	30	GTCS	febrile	cns inf	2	bus	nil	nil	n	yes	no	99	no	no	no	1. DI	YES	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
15	Priyan	84	6-10	m	self	0.5	60	GTCS	febrile	cns inf	2	bike	nil	nil	n	yes	no	97	no	h	no	3. P1	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
16	Anad periyaswamy	132	> 10	m	gh	8.0	30	GTCS	afebrile	all	2	amb	nil	nil	n	yes	no	95	no	no	no	6. M1	YES	NO	NO	-	-	-	TUMOUR	AMA
17	Aakash	21	1- 5	m	self	0.8	40	GTCS	febrile	NO RISK	2	amb	nil	nil	n	yes	y	96	no	no	no	5. PH	NO	NO	NO	-	n	-	IDIOPATHIC S D	RECOVERED
18	Kayamydeen	24	1- 5	m	self	50.0	60	GTCS	febrile	asd/lri	2	amb	nil	nil	n	yes	no	97	no	no	no	2. D2	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
19	Kasamadhan	36	1- 5	m	self	0.5	30	GTCS	febrile	dd	1	amb	nil	nil	y	yes	no	94	no	no	no	3. P1	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
20	Loganathan	48	1- 5	f	self	5.0	40	GTCS	febrile	fever	1	amb	yes	diazepam	n	yes	no	90	no	h	no	2. D2	YES	YES	YES	-	a	-	REMOTE CAUSE	RECOVERED-S
21	Balashankar	72	6-10	m	gh	6.0	35	focal-s	afebrile	NO RISK	1	amb	nil	nil	n	yes	no	93	no	h	no	1. DI	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED

S.NO	Name	AGE	AGE.GROUP	SEX	REFFROM	DISTANCE	DURAFIT	FITTYPE	FEBAFEB	RISKFACT	neurol.nor.abnor	TRANSP	PREHOSP	DRUG	PASTHO	BVM	INTUBATI	SAO2	SHOCK	GLUCOSE	CALCIUM	AED	ACIDOSIS	ICP	deficit	LP	CTBRAIN	MRI	FINALDIAGNOSIS	outcome
22	Indu	120	6-10	f	self	0.8	30	GTCS	febrile	known sd	2	amb	nil	nil	y	yes	no	92	no	no	no	3. P1	NO	NO	NO	-	n	-	IDIOPATHIC S D	RECOVERED
23	Diana epsiba	10	< 1	f	pvt	8.0	35	GTCS	febrile	NO RISK	2	amb	nil	nil	n	yes	no	96	no	no	no	2. D2	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
24	Prema	120	6-10	f	self	4.0	40	GTCS	afebrile	sturge weber	1	amb	nil	nil	y	yes	no	95	no	no	no	4. P2	NO	NO	NO	-	a	a	NEUROCUTANEOUS SYNDROME	RECOVERED
25	Selvavani	12	< 1	m	pvt	23.0	30	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	94	no	no	no	3. P1	YES	NO	YES	-	a	a	IDIOPATHIC S D	RECOVERED
26	Madhani	96	6-10	m	gh	6.0	30	GTCS	afebrile	leukodystrophy	1	amb	nil	nil	y	yes	no	96	no	no	no	2. D2	YES	NO	NO	-	-	a	I E METABOLISM	RECOVERED
27	Muthusudali	108	6-10	m	self	4.0	60	GTCS	afebrile	known sd	2	amb	nil	nil	y	yes	no	95	no	no	no	3. P1	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
28	Sumaiyabanu	60	1- 5	m	self	5.0	30	GTCS	febrile	fever	2	car	nil	nil	n	yes	no	99	no	no	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
29	Mahendrakumar	48	1- 5	m	self	0.5	35	GTCS	febrile	NO RISK	2	amb	nil	nil	n	yes	no	90	no	no	no	2. D2	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
30	Muthukannan	120	6-10	m	gh	0.8	45	GTCS	afebrile	dd	1	amb	nil	nil	y	yes	no	97	no	no	no	3. P1	NO	NO	NO	-	-	-	REMOTE CAUSE	EXPIRED
31	Chinnathambi	8	< 1	m	self	9.0	35	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	94	no	no	no	3. P1	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
32	Muthumani	120	6-10	m	gh	12.0	32	GTCS	afebrile	dd	1	amb	yes	diazepam	y	yes	no	92	no	no	no	2. D2	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
33	Essakiraj	72	6-10	m	self	40.0	240	GTCS	febrile	cns inf	2	auto	nil	nil	n	yes	y	84	y	no	no	9. M4	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	EXPIRED
34	Prathiba	108	6-10	f	self	25.0	40	NCSE	febrile	cns inf	2	amb	nil	nil	n	yes	no	95	no	no	no	3. P1	NO	NO	NO	-	a	-	NON COMPLIANCE	RECOVERED
35	Muthukannan	11	< 1	m	pvt	12.0	120	GTCS	afebrile	withdrawal	2	amb	yes	diazepam	y	yes	no	96	no	no	no	2. D2	NO	NO	NO	-	-	-	NON COMPLIANCE	RECOVERED
36	Murugeswari	11	< 1	f	gh	20.0	35	GTCS	febrile	cns inf	2	bus	yes	diazepam	n	yes	no	96	no	no	no	3. P1	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
37	Saravan	18	1- 5	m	gh	50.0	45	GTCS	febrile	fever	2	bike	yes	phenytoin	n	yes	no	93	no	no	no	2. D2	NO	NO	NO	-	n	-	FEBRILE SEIZURE	RECOVERED
38	Aswathi	12	< 1	f	gh	25.0	120	GTCS	febrile	trauma	2	amb	yes	diazepam	n	yes	no	92	no	h	no	4. P2	NO	NO	NO	-	n	-	TUMOUR	RECOVERED
39	Ramalakshmi	96	6-10	f	phc	45.0	300	NCSE	febrile	withdrawal/ abscess	1	amb	yes	diazepam	y	yes	y	87	no	no	no	8. M3	YES	NO	YES	-	-	a	NON COMPLIANCE	EXPIRED
40	Kiruba	96	6-10	f	self	50.0	240	GTCS	febrile	cns inf	2	amb	yes	diazepam	n	yes	no	91	no	no	no	3. P1	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED - S
41	Viyalakshmi	72	6-10	f	pvt	25.0	60	GTCS	afebrile	dd	1	amb	nil	nil	y	yes	y	91	no	no	no	5. PH	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
42	Manisheela	5	< 1	f	phc	32.0	30	GTCS	febrile	dd/cp	1	amb	nil	nil	y	yes	no	97	no	no	no	3. P1	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
43	Kangaraj	72	6-10	f	self	12.0	30	GTCS	febrile	sd	2	amb	nil	nil	y	yes	no	96	no	no	no	2. D2	NO	YES	NO	-	-	-	IDIOPATHIC S D	RECOVERED

S.NO	Name	AGE	AGE.GROUP	SEX	REFFROM	DISTANCE	DURAFIT	FITTYPE	FEBAFEB	RISKFACT	neurol.nor.abnor	TRANSP	PREHOSP	DRUG	PASTHO	BVM	INTUBATI	SAO2	SHOCK	GLUCOSE	CALCIUM	AED	ACIDOSIS	ICP	deficit	LP	CTBRAIN	MRI	FINALDIAGNOSIS	outcome
44	Maharasi	5	< 1	f	self	12.0	45	GTCS	febrile	cns inf/septic shock/rf	2	amb	nil	nil	n	yes	y	80	y	l	no	2. D2	YES	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
45	Darshini	60	1- 5	f	self	30.0	480	GTCS	febrile	cns inf	1	amb	nil	nil	n	yes	no	94	y	no	no	6. M1	YES	NO	NO	-	-	-	REMOTE CAUSE	EXPIRED
46	Esakkimuthu	33	1- 5	m	self	20.0	30	GTCS	febrile	fever	2	car	nil	nil	n	yes	no	93	no	no	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
47	Sheeba	120	6-10	f	self	7.0	30	GTCS	febrile	dd	1	bus	nil	nil	y	yes	no	92	no	no	no	1. DI	NO	YES	NO	-	a	-	REMOTE CAUSE	RECOVERED
48	Agitha	132	> 10	f	gh	60.0	240	GTCS	afebrile	res fail/shock/trauma	1	amb	yes	phenytoin	n	yes	y	85	y	l	no	5. PH	NO	NO	NO	-	-	-	REMOTE CAUSE	EXPIRED
49	Mariya deepa	48	1- 5	f	phc	32.0	30	GTCS	afebrile	withdrawal	1	amb	nil	nil	y	yes	no	94	no	no	no	6. M1	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
50	Shakthi	36	1- 5	f	self	0.5	30	GTCS	febrile	dd	1	bus	nil	nil	y	yes	no	93	no	l	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
51	Swetha	96	6-10	f	self	0.8	35	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	96	no	no	no	2. D2	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
52	Manibarathy	108	6-10	m	gh	3.0	45	GTCS	afebrile	sd	2	amb	nil	nil	y	yes	no	96	no	no	no	3. P1	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
53	Yesuraja	12	< 1	m	gh	30.0	30	GTCS	febrile	dd/fever	1	amb	nil	nil	y	yes	no	96	no	l	no	3. P1	NO	YES	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
54	Subithra	3	< 1	f	self	30.0	45	FOCAL	afebrile	cns hage	2	amb	nil	nil	n	yes	no	94	no	no	no	6. M1	NO	NO	NO	-	a	a	CNS HEMORRHAGE	RECOVERED - S
55	Kandaiselvi	21	1- 5	f	gh	10.0	30	GTCS	febrile	fever	2	amb	nil	nil	n	yes	no	94	no	no	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
56	Selvi	36	1- 5	f	self	0.5	40	GTCS	febrile	aspiration	2	amb	nil	nil	n	yes	y	90	no	no	no	2. D2	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
57	Veerapandian	48	1- 5	m	pvt	24.0	35	GTCS	febrile	dd	1	amb	nil	nil	y	yes	no	97	no	no	no	1. DI	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
58	Essakimuthuraman	84	6-10	m	pvt	7.0	30	GTCS	afebrile	NO RISK	2	amb	nil	nil	n	yes	no	93	no	no	no	2. D2	NO	NO	NO	-	a	-	IDIOPATHIC S D	RECOVERED
59	Krishnamoorthy	132	> 10	m	gh	42.0	45	GTCS	febrile	cns inf	1	amb	yes	phenytoin	n	yes	y	93	no	l	no	5. PH	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED
60	Preethika	8	< 1	f	self	23.0	35	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	94	no	no	no	2. D2	YES	NO	NO	n	n	-	ACUTE CNS INFECTION	RECOVERED
61	Bathrakali	3	< 1	m	gh	6.0	45	GTCS	febrile	cns inf/septic shock/rf	2	amb	yes	pheno	n	yes	y	86	y	no	no	5. PH	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
62	Mariadeepa	48	1- 5	f	self	50.0	35	GTCS	febrile	cp	1	amb	nil	nil	y	yes	no	93	no	l	no	1. DI	YES	NO	NO	-	-	-	REMOTE CAUSE	EXPIRED
63	Durgadevi	48	1- 5	f	self	20.0	180	GTCS	febrile	cns hage	2	amb	nil	nil	n	yes	no	95	no	h	no	4. P2	NO	NO	NO	-	a	a	CNS HEMORRHAGE	RECOVERED
64	Ponmadaswamy	96	6-10	m	self	15.0	35	GTCS	febrile	NO RISK	2	amb	nil	nil	n	yes	no	96	no	no	no	2. D2	YES	NO	NO	-	-	-	IDIOPATHIC S D	EXPIRED
65	Karthikannan	72	6-10	m	self	45.0	45	GTCS	febrile	fulminant hepatitis	2	amb	nil	nil	n	yes	no	95	no	no	no	2. D2	NO	YES	NO	-	a	-	SYSTEMIC ILLNESS	RECOVERED

S.NO	Name	AGE	AGE.GROUP	SEX	REFFROM	DISTANCE	DURAFIT	FITTYPE	FEBAFEB	RISKFACT	neurol.nor.abnor	TRANSP	PREHOSP	DRUG	PASTHO	BVM	INTUBATI	SAO2	SHOCK	GLUCOSE	CALCIUM	AED	ACIDOSIS	ICP	deficit	LP	CTBRAIN	MRI	FINALDIAGNOSIS	outcome
66	Manju	11	< 1	f	self	12.0	35	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	y	88	no	no	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
67	Nishanthipriya	84	6-10	f	self	6.0	40	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	96	no	h	no	2. D2	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
68	Gowri	36	1- 5	f	gh	32.0	35	GTCS	febrile	fever	2	amb	nil	nil	n	yes	y	90	no	h	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
69	Vinojayaraj	72	6-10	m	phc	25.0	30	GTCS	febrile	pms	1	amb	nil	nil	y	yes	no	96	no	no	no	4. P2	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
70	Radha	72	6-10	f	self	12.0	45	GTCS	afebrile	withdrawal	1	amb	nil	nil	y	yes	no	97	no	no	no	3. P1	YES	NO	NO	-	-	-	REMOTE CAUSE	EXPIRED
71	Gaythri	36	1- 5	f	gh	45.0	480	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	y	88	no	l	no	9. M4	NO	NO	NO	a	n	-	ACUTE CNS INFECTION	RECOVERED
72	Helenanushya	24	1- 5	f	self	8.0	45	GTCS	afebrile	NO RISK	2	amb	nil	nil	n	yes	no	96	no	h	no	9. M4	NO	NO	NO	-	-	-	IDIOPATHIC S D	RECOVERED
73	Madaswamy	48	1- 5	m	self	12.0	30	GTCS	febrile	lri	2	amb	nil	nil	n	yes	no	97	no	h	no	1. DI	NO	NO	NO	-	-	-	SYSTEMIC ILLNESS	RECOVERED
74	Karthikannan	48	1- 5	m	self	25.0	40	GTCS	febrile	dd/cns inf	1	amb	nil	nil	y	yes	no	94	no	no	no	2. D2	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
75	Mahalakshmi	24	1- 5	f	self	25.0	35	GTCS	febrile	fever	2	amb	nil	nil	n	yes	no	94	no	no	no	1. DI	YES	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
76	Veerapandian	54	1- 5	m	pvt	4.0	30	GTCS	febrile	dd	1	amb	yes	diazepam	y	yes	no	93	no	no	no	4. P2	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED
77	Hema	120	6-10	f	gh	21.0	45	GTCS	febrile	migraine	2	amb	yes	diazepam	n	yes	no	98	no	no	no	1. DI	NO	NO	NO	-	a	-	IDIOPATHIC S D	RECOVERED - S
78	Lakshmi	8	< 1	f	self	35.0	45	GTCS	febrile	add/adem	2	amb	nil	nil	n	yes	y	99	no	no	no	5. PH	NO	NO	NO	n	n	-	ACUTE CNS INFECTION	RECOVERED
79	Karthik	96	6-10	m	self	25.0	35	focal-s	afebrile	dd/cp	1	amb	nil	nil	y	yes	no	96	no	no	no	3. P1	NO	NO	YES	-	a	-	REMOTE CAUSE	RECOVERED - S
80	Thalavai	132	> 10	m	pvt	34.0	180	FOCAL	afebrile	dd	1	amb	yes	diazepam	y	yes	no	94	no	no	no	3. P1	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED
81	Ponpandi	132	> 10	m	self	7.0	30	GTCS	febrile	cns inf	2	amb	nil	nil	n	yes	no	90	no	no	no	3. P1	NO	NO	NO	a	a	-	ACUTE CNS INFECTION	RECOVERED
82	Ramlakshmi	36	1- 5	f	self	25.0	35	NCSE	febrile	lri	2	amb	nil	nil	n	yes	no	91	no	no	no	3. P1	NO	NO	NO	n	a	-	SYSTEMIC ILLNESS	RECOVERED
83	Sofya	24	1- 5	f	self	35.0	38	GTCS	afebrile	hemiplegia	1	amb	nil	nil	y	yes	no	92	no	no	no	6. M1	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED
84	Shanmugaraja	12	< 1	m	self	25.0	30	FOCAL	febrile	fever	2	amb	nil	nil	n	yes	no	91	no	no	no	1. DI	NO	NO	NO	-	-	-	FEBRILE SEIZURE	RECOVERED
85	Anush	12	< 1	m	pvt	15.0	35	GTCS	febrile	dd	1	amb	yes	diazepam	y	yes	no	90	no	h	no	2. D2	NO	NO	NO	n	-	-	FEBRILE SEIZURE	RECOVERED
86	Maharajan	30	1- 5	m	self	14.0	120	GTCS	afebrile	cp	1	amb	yes	phenytoin	y	yes	no	92	no	no	no	7. M2	NO	NO	NO	-	a	-	REMOTE CAUSE	RECOVERED
87	Gopalakrishan	48	1- 5	m	self	6.0	45	FOCAL	febrile	dd	1	amb	yes	diazepam	y	yes	y	94	no	no	no	5. PH	NO	NO	NO	-	a	-	NEUROCUTANEOUS SYNDROME	RECOVERED

S.NO	Name	AGE	AGE.GROUP	SEX	REFFROM	DISTANCE	DURAFIT	FITTYPE	FEBAFEB	RISKFACT	neurol.nor.abnor	TRANSP	PREHOSP	DRUG	PASTHO	BVM	INTUBATI	SAO2	SHOCK	GLUCOSE	CALCIUM	AED	ACIDOSIS	ICP	deficit	LP	CTBRAIN	MRI	FINALDIAGNOSIS	outcome
88	Iswarya	84	6-10	m	self	25.0	50	GTCS	afebrile	withdrawal	2	amb	nil	nil	y	yes	no	92	no	no	no	3. P1	NO	NO	NO	-	-	-	NON COMPLIANCE	RECOVERED
89	Ulaganathan	72	6-10	m	self	15.0	60	NCSE	afebrile	dd/cp	1	amb	nil	nil	y	yes	no	94	no	no	no	2. D2	NO	NO	NO	-	a	a	NON COMPLIANCE	RECOVERED
90	Essakimuthuraman	108	6-10	m	gh	6.0	60	GTCS	afebrile	cp	1	amb	yes	diazepam	y	yes	no	92	no	h	no	6. M1	NO	NO	NO	-	-	-	REMOTE CAUSE	RECOVERED
91	Manikandan	132	> 10	m	gh	8.0	30	GTCS	afebrile	all	2	amb	nil	nil	n	yes	no	95	no	no	no	6. M1	YES	NO	NO	-	-	-	TUMOUR	AMA
92	Loothu	84	6-10	f	self	7.0	35	MULTI	afebrile	NO RISK	2	bike	nil	nil	n	yes	no	96	no	no	no	4. P2	NO	NO	NO	-	-	-	IDIOPATHIC S D	AMA

Abbreviations

M	Male
F	Female
CP	Cerebral Palsy
DD	Development Delay
amb	Ambulance
y	yes
n	no
D1	Diazepam first dose
D2	Diazepam Second dose
P1	Phenytoin first dose
P2	Phenytoin second dose
PH	Phenobarbitone
M1	Midazolam 1microgram / kg / min
M2	Midazolam 2microgram / kg / min
M3	Midazolam 3microgram / kg / min
M4	Midazolam 4microgram / kg / min
gh	Government Hospital
pvt	Private Hospital
phc	Primary Health Centre
CNS inf	Central Nervous system Infection
a	abnormal
n	normal
1	Neurologicaly abnormal
2	Neurologically normal
Recovered - S	Recovered with Sequele